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TISSUE CHANGES IN THE NASAL MUCOSA. PRELIMINARY REPORT.*

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The nasal mucosa furnishes an excellent source of material for the study of a large variety of microscopical tissue changes and for the correlation of these changes with the manifold signs and symptoms that are prevalent in human beings. There is fortunately a heterogeneity of structure in this organ which results in a diversified interplay of the various components as the organ responds to various stimuli. Since innumerable opportunities present themselves to secure fresh specimens of nasal mucous membrane in the living, it is obvious that here there is afforded an important means of obtaining evidence concerning cellular adaptive and protective responses to different types of stimuli and reflexes.

During the past seven years such an approach and study of the nasal mucosa has been made possible by the co-operation, guidance and stimulation of Dr. Harris P. Mosher and Dr. Frederick H. Verhoeff, under the auspices of the Massachusetts Eye and Ear Infirmary, Massachusetts General Hospital and Harvard Medical School. Specimens of the nasal mucosa were obtained in the various stages of rest, congestion, early rhinitis, subacute and chronic conditions, polyp formations, vasomotor and anaphylactic conditions. The microscopical pictures were carefully correlated with the patient's subjective symptoms, with the objective appearance of the mucosa as seen by the examiner, and with the subsequent developments in the patient's condition. The subject-matter of this paper is confined

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chiefly to the microscopical findings. Various conclusions and further experimentation that have resulted from the correlation of all these observations are being prepared in greater detail.

The average nasal mucosa contains a certain relative amount of connective tissue, lymphocytes, other lymphoid cells, eosinophiles, blood vessels, cavernous vessels, glands lymphatics and distended interstitial spaces. In definitely abnormal states the nasal mucosa presents chiefly relative increase or decrease in these tissue elements under the epithelium, that is, in the tunica propria or stroma. In the majority of conditions to be discussed here the epithelium shows relatively little change. Likewise the underlying periosteum, perichondrium, cartilage and bone are of secondary moment for our present purposes. Changes in the bone have always appeared inconspicuous even in the marked pathological conditions of the nasal mucosa. It might also be stated that the olfactory elements, which are confined to the upper third of the nasal mucosa, are not considered here.

For a concise, clear description of the components of the average nasal mucosa, the following extract is quoted from Piersol's Human Anatomy, 1919 edition, page 1416:

"The mucous membrane of the respiratory region differs greatly in thickness in various parts of the nasal fossa. In situations where the contained cavernous tissue is well represented, as over the inferior turbinate, it may reach a thickness of several millimeters, while when such a tissue is wanting, as on the lateral wall, it is reduced to less than a millimeter.

"The epithelium is stratified ciliated columnar in type and includes the tall surface cells, bearing the cilia, between the inner ends of which lie irregularly columnar basal cells. Numerous elements exhibit various stages of conversion into mucous-containing goblet cells. The current produced by the cilia is toward the posterior nares.

"Beneath the epithelium stretches the membrana propria, or basement membrane, that varies greatly in thickness; although in certain localities feebly developed, it is usually well marked. Under pathological conditions its thickness may increase fourfold or more. In many places the membrana propria is pierced by minute vertical channels, the basal canals, in which connective tissue cells and leucocytes are found, but never blood capillaries (Schiefferdecker).

"The tunica propria consists of interlacing bundles of fibro-elastic tissue which are most compactly disposed towards the subjacent periosteum. The looser superficial stratum is rich in cells and here and there contains aggregations of lymphocytes that may be regarded as masses of adenoid tissue (Zuckerkandl). In certain parts of the nasal fossa the stroma of the mucous membrane contains vascular areas

composed of numerous intercommunicating blood-spaces that confer the character of a true cavernous tissue. These specialized areas, the corpora cavernosa, as they are called, are especially well developed over the inferior and lower margin and posterior extremity of the inferior and middle turbinates, and less so over the posterior end of the upper turbinate and tuberculum septi. When typical they occupy practically the entire thickness of the mucous membrane from periosteum to epithelium, the interlacunar trabeculae containing the glands and blood vessels destined for the subepithelial stroma. The blood sinuses, the general disposition of which is vertical to the bone (Zuckerkandl), include a superficial reticular zone of smaller spaces and a deeper one of larger lacunae. The engorgement and emptying of the cavernous tissue is controlled by nervous reflexes and probably has warming of the inspired air as its chief purpose (Kallius).

"The glands of the respiratory region are very numerous, although varying in size, tuboalveolar in form, and, for the most part, mixed mucous in type. The chief ducts open on the free surface by minute orifices barely distinguishable with the unaided eye. Their deeper ends branch irregularly into tubes that bear the ovoid terminal alveoli. The latter are lined with mucous secreting cells, between which lie the rescentic groups of serous cells that stamp the glands as mixed (Stohr). Exceptionally exclusively serous glands are also encountered (Kallius)."

ACUTE NASAL CONGESTIONS.

Under this heading are included the great variety of conditions, ranging from mild short, transitory attacks to the more persistent ones that continue for several days or go on to develop later into the common typical purulent head cold.

At the onset a "cold" which later turns out to be very mild and which never gets beyond the watery stage to the purulent state cannot apparently be positively distinguished from a "cold" which may progress to purulent exudation and purulent sinusitis. Both types may present a similar pink swollen nasal mucosa with varying degrees of watery discharge, sneezing and subjective sensations of discomfort.

Microscopically a specimen of a swollen mucosa at this stage shows in the stroma an edema, an increase in the mononuclear lymphoid cells, and a definite increase in eosinophiles. The eosinophiles are more abundant near the periphery under the epithelium. In some cases a marked dilatation of the cavernous blood vessels in the turbinates was found; in others, the state of the cavernous vessels seemed to be within the limit of variation which is often found in uncongested turbinates. However, it must be borne in mind that it is possible for a marked collapse or alteration in the state of the deli-

cate cavernous vessels to occur at the moment the tissue specimen is removed. Additional data is, of course, desirable on this point.

In such specimens are found no polymorphonuclear neutrophilic leucocytes, no observable dilatation of the ordinary arteries or veins, no abnormal increase of the lymphoid cells or eosinophiles within the lumen of any arteries and veins, and no ordinary bacteria are demonstrable under the epithelium with the Gram stain method. Thus none of the classical signs of bacterial infection are apparent in the nasal mucosal tissue in this early change, which finding is contrary to a widespread assumption today.

This same microscopical reaction and picture was also consistently demonstrated to occur in patients who were prone to rapid congestion of the nose following shortly after an exposure to chilling or to a draught. These congestions were usually of short duration and often alternated within short periods of time from one side of the nasal fossa to the other and then might vanish completely or continue along and in a few days result in a typical purulent head cold. The rapidity with which these nasal blockings, watery discharges and sneezing spells appear and disappear in people has led to the obvious deduction or feeling that the early symptoms are the result of a nerve reflex from the skin to the nasal mucosa. This type of reflex is commonly classed in the vasomotor group. To ascribe these rapid clinical manifestations and rapid mutation of symptoms to bacteria would obviously be inconsistent with most of our notions and facts concerning bacterial action and influence.

ACUTE PURULENT RHINITIS.

The preliminary period of watery congestion may last a varying period, usually from one to three days, before the purulent nasal discharge appears. With this change the feeling of oppressive congestion in the nose is as a rule relieved. However, there may be temporary less severe recurrences of the subjective feeling of blocking in the nose during the next few days.

Here the polymorphonuclear neutrophilic leucocytes and dilated capillaries containing them appear close under the epithelium. Many of these leucocytes, as well as other leucocytes, can be seen migrating through the epithelium toward the outer surface. There is less interstitial edema. The eosinophiles diminish in number. There is still a marked increase in lymphocytes and perhaps a hypertrophy of the occasional small lymph nodes that are scattered in the mucosa.

With the subsidence of the purulent discharge and the return of the mucosa to an average resting state, the polymorphonuclear neutrophilic leucocytes disappear completely and the occasional eosinophile reappears. The hyperplasia of lymphoid cells, however, may remain

in more or less degree. If a patient has repeated purulent colds, there is apt to result a permanent relative increase in the following lymphoid cells: *1.* small round cells, the lymphocytes, which have densely staining nuclei and scanty cytoplasm; *2.* plasma cells which tend to appear in repeated, subacute, or chronic conditions of this sort; *3.* large, more or less round cells which contain a small, centrally-located, shrunken pyknotic nucleus; the cytoplasm of these large cells is abundant, appears swollen, and stains homogeneously either as pale blue or pale pink with the usual hematoxylin and eosin stain method; numerous forms that appear to be transitional stages from plasma cells to the large cells are frequently present.

CHRONIC PURULENT RHINITIS.

This class varies from the subacute groups that have remissions and tend to recur quite regularly on exposure to unfavorable atmospheric conditions and other irritative stimuli to the chronic states that constantly have a purulent nasal discharge. A hypertrophic type of tissue reaction is usually found in these situations.

Here the microscope reveals a definite increase in lymphoid elements and in amount of connective tissue. The eosinophiles are absent or very meager in number. The prominent lymphoid elements are usually small round cells and plasma cells. The plasma cells seem to appear consistently in the chronic conditions. The polymorphonuclear neutrophilic leucocytes vary considerably in the tissue, from very small numbers to large numbers. Here also the polymorphonuclear leucocytes seem more abundant and appear first near the epithelium. It is often very striking to find a remarkably small number of these leucocytes in cases which clinically are exudating large amounts of purulent material daily over a period of years.

Many degenerative cells and apparently transitional types of cells are seen at times. These cells are usually of the large lymphoid variety, containing small nuclei and abundant cytoplasm.

Even in the extreme conditions of purulent infection encountered it is significant that the presence of bacteria in the tissues is usually difficult to demonstrate. The same pathological tissue changes are encountered in cases of undoubted osteomyelitis, such as is seen in extensive ethmoidal cell degeneration or a direct involvement of bone from an apical tooth abscess leading into the maxillary antrum. Thickened or polypoid mucous membrane removed from such areas may show a marked infiltration with lympocytes and plasma cells, hyperplasia of connective tissue, and increased glandular activity with gland inclusions but little or no polymorphonuclear neutrophilic leucocytes, bacteria or hyperemia.

VASOMOTOR RHINITIS.

A great many people have frequent recurrent "colds" and they are given the impression that they are suffering with chronic nasal infections, often more or less dependent on anatomical or structural defects. However, on questioning, they admit that their colds hardly ever get purulent, but consist of sudden onset with a lot of sneezing, blocking of a changeable type and watery discharge. This train of symptoms may last a few hours to a week or more. If they do get a real purulent head cold, they notice that they are usually free of the minor colds for a month or two after that. This is the great group that has been classified as vasomotor rhinitis.

Reference has already been made to the microscopical picture found in the mild vasomotor type. The more severe types are essentially similar in their tissue change. The outstanding pathological features are the edema and the marked increase in tissue eosinophiles, the latter being more numerous near the epithelium. There seems to be no observable hyperemia or increase in the eosinophile cells in the arteries and veins. As the condition persists, the small round cells and plasma cells in the stroma begin to increase in number as compared with the number of eosinophiles. The number of polymorphonuclear neutrophilic leucocytes is negligible. There is no excessive proliferation of connective tissue.

In severe seasonal vasomotor conditions, for example, in the typical ragweed hay-fever cases, the same tissue changes are evident. The numbers of eosinophiles are astonishing, being more abundant nearer the epithelium. There may be mononuclear eosinophiles as well as polymorphonuclear eosinophiles. The interstitial edema is marked. The cavernous vessels are usually found dilated. The glands usually show signs of great activity.

When the mucosa over a turbinate exhibits a rapid increase or decrease in volume, the important change apparently is in the size of the cavernous vessels. Thus the congested pink-looking turbinate appears and subsides. There is also of frequent occurence, however, the pale swollen boggy-looking turbinate with varying degrees of bluish hue in it. This is more apt to be found in more severe seasonal types of vasomotor cases. Microscopically, this pale tissue presents a marked increase in interstitial edema with relatively less dilatation of vessels. Clinically, changes in volume of such turbinates seem to require more time than those of a pinker hue.

It is also of great interest and importance to examine the mucous membrane in vasomotor cases during clinically quiescent periods, that is, at the so-called interval period between disturbances. Macroscopically, the mucosa may appear like any average nasal membrane

or may have more or less localized or generalized thickenings. Microscopically, a consistent definite change is noted as compared with conditions present in the average nasal mucosa. Relatively a far greater number of eosinophiles is present. There are more apt to be an increased number of areas of localized edema and increased lymphoid infiltration, such as lymphocytes, plasma cells and large lymphoid cells. Changes of this nature were demonstrable in the mucosae of hay-fever patients even years after they had stopped having attacks of hay-fever.

Of extremely great importance is recognition of the fact that a purulent bacterial infection can be superimposed upon a nasal mucosa during vasomotor attacks. When the vasomotor upset is mild or of short duration, the purulent discharge is apt to run a certain definite cycle and clear up, as has been pointed out in the discussion of acute nasal congestions. When the upsetting vasomotor condition is prolonged or frequently recurrent, the secondary bacterial infection often obscures, by its purulent manifestations, the underlying vasomotor conditions at the time of clinical examination or surgical operation. Hence these cases are apt to be the obstinate and recurrent ones which do not clear up in spite of what is considered the most careful and competent therapy.

The microscopic picture of this last group of cases is identically the same as that already described under the heading of chronic purulent rhinitis. The interplay between lymphocytes, plasma cells and polymorphonuclear leucocytes associated with some proliferation of connective tissue presents a condition indistinguishable from that seen in the average case of chronic purulent rhinitis or sinusitis. The thickened hypertrophic membranes which are found in chronic or recurrent vasomotor rhinitis with superimposed purulent infection on the one hand, and in chronic purulent rhinitis on the other, seem to be similar and alike in their various manifestations and variations as seen under the microscope.

NASAL POLYPS.

The mechanism of the formation of nasal polyps will not be discussed here, other than to intimate that it is probable that an important factor which concerns their formation and localization is the uneven distribution of the intensity of an underlying vasomotor disturbance in various parts of the nasal mucosa. Following the method adopted in the foregoing descriptions of the various changes which occur in the nasal mucosa, it will clarify matters considerably if a few words are said about the clinical occurrences of polyps. The reader may be enabled to visualize more clearly the clinical appear-

ance and relationships of the various types of polyps described.

Polyps described here vary in size and consistency from small areas of transitory localized edema to permanent, more or less firm tumor masses. They have been found to occur in association with purulent discharge and with repeated nasal congestions as well as in noses where there apparently has been no appreciable or obvious past or present nasal purulent discharge or any generalized subjective feelings of congestion on the part of the patient. The site of origin or attachment of these polyps and polypoid conditions is most frequently found to be the nasal sinuses, the turbinates, the middle and superior meati.

The current opinion today seems to associate polyp formation in the nose with bacterial etiology and infers that the mere presence of polyps suggests that there has been purulent infection and discharge. It is remarkable to find, fairly often, patients with many nasal polypi insisting that they have never had any purulent nasal discharge and even rarely have a severe watery nasal congestion. In spite of this history, numerous polyps may be seen in the ethmoids, antrum, and even extending into the postnasal space. Likewise, so many vasomotor rhinitis patients and asthmatic patients, with or without nasal vasomotor symptoms, present nasal polyps. These polyps, further, tend to recur and recur in spite of surgical removal and without any obvious coexisting purulent signs of infection. It is also paradoxical to see patients who have profuse purulent discharge for years show no tendency to polyp formation or to blocking of any of the ostia or ducts of the sinuses. On the other hand, a patient will appear with a history of repeated very mild nasal congestions with little or no purulent discharge, and his nose may present one or all the sinuses full of polyps. Furthermore, these polyps seem very often to be causing no discomfort or disturbance. It might be mentioned here that many patients often feel a blocking in their nose as if they had polyps obstructing their attempts at inspiration and expiration of air, but inspection reveals merely a diffuse slight congestion or thickening of the entire nasal mucosa; this group seems to have a tendency to develop polyps if the condition persists over a long period.

Microscopically, the early stages of a nasal polyp is usually manifested in the stroma by a localized increase of interstitial edema, lymphoid infiltration, *i.e.,* chiefly lymphocytes and plasma cells, and an increase in the number of eosinophiles. As the polyp persists there is a proliferation of young connective tissue cells and invasion by some capillaries and small arterioles. Apparently then the same types of tissue reactions and responses are being dealt with in studying

nasal polyps as in studying more diffuse changes in the nasal mucosa, as outlined above.

The same interplay of vasomotor reflexes and factors with superimposed purulent infections are found to have corresponding influences on the further development of the polyps. It is surprising to see fairly large polyps recede spontaneously, probably due to the cessation of the underlying stimulation or irritation.

In cases where polyps appear during the course of a severe vasomotor irritation, as in typical hay-fever, the polyp contains a striking number of eosinophiles and marked areas of edema. The eosinophiles here are also more abundant near the periphery of the polyp under the epithelium. It is difficult to determine whether the edema is mostly due to a distention of pre-existing lymph spaces, or to a distention of other tissue spaces or to an increased permeability of the capillaries, allowing lymph and serum into the general tissue spaces. A polyp started this way may disappear or persist after the attack of hay-fever is over. Connective tissue proliferates in varying amount throughout the polyp if it persists and grows older. Lymphocytes and plasma cells may increase in numbers relatively. The base of the polyp becomes pedunculated and considerable fibrous connective tissue may be laid down in this region, *i.e.*, the base and neck of the polyp. This connective tissue hyperplasia is true of most all polyps that persist for some time.

Polyps which occur in nasal mucosae that are subject to perennial types of vasomotor disturbances or which occur in vasomotor conditions associated with asthma, present the same picture as has just been described as occurring in the case of hay-fever. The average polyp removed from the nasal sinuses or middle meatus of one of these patients will show a marked infiltration with eosinophiles associated with edema, some round cell infiltration and connective tissue formation. If a secondary purulent infection is present, the plasma cells increase in number and the eosinophiles diminish. As the purulent exudation becomes more marked clinically, polymorphonuclear neutrophilic leucocytes appear under the epithelium and may spread deeper through the polyp stroma, but they are always more abundant nearer the epithelium. In these late stages and in the stationary states that polypoid conditions often assume, the large lymphoid cell with the small pyknotic nucleus and abundant pale staining homogeneous cytoplasm often appears in increased numbers.

CYSTIC POLYPOID FORMATIONS.

Under this heading three distinct subdivisions will be included, *viz.*, mucocele formation, mesothelial cysts and cystic degeneration of polyps.

A mucocele of a whole nasal sinus occurs when the ostium of the sinus is blocked and the secreting cells continue to secrete mucus. This material collects and fills the sinus, let us say the maxillary antrum, the frontal sinus, or a single occluded ethmoid cell. As the pressure accumulates it may erode adjacent bony structures, or reach a standstill, perhaps due to the pressure of the collected mucus causing atrophy of the lining epithelial cells, or the mucus material may become purulently infected.

There may be local small mucocele formations in a nasal sinus. This is quite common in the maxillary antrum. The outlet of a gland may become blocked off due to a hypertrophic process in the mucosa. Thus, several polypoid masses attached to various walls may be present at one time in the antrum without any involvement of the normal ostium. These are inclusion cysts with true epithelial linings and are often present in polyps. The mucus contained in them may vary from clear mucus containing various epithelial cell debris and leucocytes, to white or yellow material, depending on the admixture of infection. They are opaque to transillumination by electric light and also are opaque to X-ray similar to ordinary polyps or pus. Beneath the epithelial lining the stroma presents a hyperplasia of connective tissue with lymphocytic, plasma cell, and large lymphoid cell infiltration.

Another type of fluid accumulation is found in the maxillary antrum fairly often. It seems to be associated symptomatically with minor vasomotor nasal congestions. This is an accumulation within the tissue spaces that causes a local mounding up or distention of the mucosa that may go on very slowly to partially or completely fill the whole antrum cavity and even cause marked thinning of the bony antral walls. There seems to be very little discomfort associated with this process. On transillumination of the sinus with electric light, it is very noticeable that the light is transmitted very well; in fact, the antrum lights up more brilliantly than the average antrum containing only air. On X-ray this fluid, however, is just as opaque as mucus, pus or a polyp.

These stromal accumulations of dilute albuminous fluid tend to be surrounded by a single layer of flat cells resembling endothelium, probably mesenchymal epithelium. The name of mesothelial cyst is suggested for this condition. Sometimes there are adjacent accumulations, which may or may not coalesce. Hence, we may get the multiocular cystic formation or a large unilocular cystic mass that collapses and disappears on pricking the surface with the escape of a thin clear fluid. The stroma adjacent to these cysts contains lympho-

cytes, plasma cells, large lymphoid cells with small nuclei and connective tissue, but seems to be low in eosinophiles. These mesothelial cysts have been found within ethmoidal polyps as well as within antral polyps; they are not infrequent in the large postnasal polyps that have their attachment and origin in the maxillary antrum.

A somewhat analogous process takes place in the so-called cystic degeneration of polyps. A patient may have a mass of polyps springing, say, from his ethmoidal region, and among these polyps are some which are very pale and soft and with free fluid content. Other polyps can be seen beginning to undergo this transformation from a somewhat firmer type. The microscopical examination here shows a scarcity of eosinophiles and lymphocytes as compared with other polyps in the same region and the accumulation of fluid within the polyp in a similar fashion as just described as mesothelial cyst formation—either unilocular or multiocular.

FIBROUS POLYPOID FORMATIONS.

Occasionally a well defined polyp, even the cystic type just described, will show localized areas of fibrosis. This is sometimes evident macroscopically as a diffuse, firm pink replacement, or even as a distinct, firm nipple-like formation. This localized type of fibrosis often occurs at the distal end of the polyp just under the epithelium. Microscopically, there is a marked hyperplasia of connective tissue in this area, with a definite disappearance of all infiltrating cells.

Another distinct type of firm hypertrophy is found on the turbinates. It is most commonly manifested by an enlargement of the anterior tip of the middle turbinate, by an enlargement of the posterior tip of the inferior turbinate, or by an elongated papillary-like hypertrophy extending especially from and along the inferior border of the inferior turbinate. These hypertrophies are not necessarily associated with frequent suppurative colds, prolonged purulent exudation, or other polypoid formation in the nasal sinuses. Clinically, they are very slowly progressive and often seem to reach a certain stage and remain stationary; many rhinologists have been tempted to consider them an isolated pathological condition confined to the turbinates alone. They rarely tend to recur on complete removal. They do seem, however, to be associated with frequent mild colds and minor attacks of nasal congestion. In other words, they may be the result of repeated low grade vasomotor or reflex disturbances to the mucosa.

The microscopic examination reveals a proliferation of fibrous tissue which is more or less patchy in its distribution. In areas there are groups of lymphoid cells, large and small. There are a few poly-

morphonuclear neutrophilic leucocytes and plasma cells, and an occasional eosinophile cells. There are numerous small, thick-walled arteries surrounded by a thick zone of dense connective tissue. Cavernous vessels are present in varying states. The epithelium is usually markedly hyperplastic in many parts.

ATROPHIC CONDITIONS OF THE NASAL MUCOSA.

Atrophic conditions are fairly common. Sometimes one part of the nasal mucosa will show atrophy and at the same time another part will show more hypertrophy. Very often the onset of the atrophic process seems to date from some severe toxemia suffered by the patient. The underlying bone is often markedly involved.

Microscopically, there is a definite reduction and disappearance of lymphoid elements, eosinophiles and other infiltrating cells. The preexisting connective tissue becomes more obvious thereby and appears to be relatively increased and to become more fibrotic in character. The epithelium loses cilia and may be metamorphosed into the squamous and pavement variety. The glands may show fatty degeneration and infiltration with small monocular cells as they atrophy.

DISCUSSION.

From the foregoing presentation it is evident that the average nasal mucous membrane in human beings is not found to be a fixed stereotyped pattern. There is a wide variation in the relative amount and number of tissue components which are commonly present in healthy individuals. In other words, some mucosae show evidence more than others of having been subject to more irritative stimuli or upset states, with consequent cellular infiltration. The reactions and infiltrations observed are presumably due to attempted defensive or adaptative responses on the part of the organism and nasal mucosa. An imprint of these experiences is thus left behind. With the ordinary conditions of life necessitating frequent interactions and adjustments of the various organs of the human body to external and internal environmental conditions, it is quite natural to find that every nasal mucosa examined exhibits a varying degree of cellular infiltration and tissue changes. It is suggested here that the term "physiological inflammation" be adopted to express the varying degrees of reaction found in the nasal mucosa of apparently healthy individuals. This phrase is used for a similar purpose by R. Rossle in his observations on the omentum (R. Rossle: Verhandl d. Deutsh path. Gesellesch., 1923, xix, 18).

By using the term "physiological inflammation" in describing the microscopical state of a nasal mucous membrane, the pathologist is

able to impart his findings to others in a precise, clear manner. For example, if a mucosa is diagnosed as being one with a "low level of physiological inflammation" it implies that in its average resting state the following picture is presented: the minimum of large and small mononuclear round cells usually found, with perhaps an occasional subepithelial aggregation of lymphoid cells forming a lymph nodule; an occasional plasma cell or eosinophile; the average amount and average type of connective tissue, no excessive localized or diffuse interstitial edema; no unusual change in the arteries, veins, cavernous vessels or glands; as a rule no polymorphonuclear neutrophilic leucocytes in the stroma; and the usual migratory leucocytes which may be numerous among the epithelial elements. Clinically, this type is the one which is not subject to frequent nasal disturbances.

A diagnosis of "a high level of physiological inflammation with a predominance of lymphocytes and plasma cells" suggests the following: an increased infiltration of small, round densely-staining mononuclear cells, either diffuse or localized or both; a concomitant increase in the large lymphoid cells containing the small shrunken nuclei; more or less hyperplasia of connective tissue; the number of eosinophiles and polymorphonuclear neutrophilic leucocytes are negligible. Clinically, this type is the one which is subject to frequent or recurrent purulent rhinitis.

If there was added to the condition just described an increased infiltration or polymorphonuclear neutrophilic leucocytes, it would be the description of the picture found in most of the cases which present clinically a chronic purulent exudation. Associated secondarily with it may be found varying degrees of membrane thickening, polypoid conditions, or gland inclusions.

Another frequent finding is a high level of physiological inflammation manifested chiefly by a marked increase in eosinophiles and in areas of edema. The number of lymphocytes may be at a low level or may be moderately increased. Clinically, this type of mucosa is found in persons who are subject, especially when exposed to sudden temperature changes, to frequent transitory nasal congestions more or less associated with sneezing and watery discharge; it is also the usual type of mucous membrane that is found to be present all year around in people who are subject to severe seasonal or perennial nasal congestions of a nonpurulent character; in other words, the whole class of typical vasomotor and anaphylactic patients. This group also frequently presents varying degrees of membrane thickening, polyps and polypoid conditions, gland inclusions and mesothelial cysts.

An admixture of lymphoid cells, such as lymphocytes and plasma cells, with more or less connective tissue hyperplasia, to the microscopical picture just described, is found to occur when the clinical conditions concerned become more firmly established. As chronicity develops, the lymphoid elements often increase and eosinophiles diminish relatively. (Various degrees of this state are often referred to as hyperplastic conditions today by various authors). Then a superimposed purulent infection may occur with the appearance of polymorphonuclear neutrophilic leucocytes in the tissues which apparently seems to be associated with a corresponding diminution or disappearance of the eosinophile cells.

It seems apparent according to the foregoing data that the nasal mucosae which present a high level of physiological inflammation microscopically are the ones which clinically are subject to frequent upsets and continued disturbances of various natures. Why, then, is this not a definite expression of what is vaguely called "the lowering of resistance" in the patient? At least, when this relative tissue change is found to be present it is coincident with a standing increased susceptibility of that nasal mucosa to be subject to congestions, in fections and polypoid changes.

Since the early microscopical picture found in conditions of transitory nasal congestions due to definite body chilling is found to be essentially the same as that found in nasal congestions that go on further to develop typical head colds and purulent exudation, it suggests that the initial upset which resulted in the lowering of resistance of the mucosa may be brought about through the same mechanism in both of these situations. The typical tissue and cellular reactions which pyogenic bacteria ordinarily call forth are not apparent. Hence it may be inferred that bacteria play a secondary role in nasal infections, being enabled to become pathogenetically active only when the resistance of the nasal mucosa has been previously lowered through some previous upset. Clinically, there are abundant examples of body chillings and exposures causing reflex disturbances in mucous membranes of the upper respiratory tract, which are, obviously, the inciting cause of the ensuing pyogenic manifestations. Incidentally, this is the prevalent age-old lay explnaation of the mechanism of these conditions and it is born of centuries of experiences.

It is suggested here that repeated cumulative irritative exposures of this latter nature may result in a series of reflexes to the nasal mucosa which results in a reaction bringing about an increased state of physiological inflammation in the tissues. Then minor exposures and reflexes can start severe upsets and make possible marked lowering of resistance to bacteria, chemicals, proteins and other sub-

stances. It might be briefly mentioned also that it is a well known observation that frequent irritative exposures will eventually result in a far greater upset in the nasal mucosa of one person than it will in another; in other words, the hereditary factor must also be basically considered when speaking of the lowering of resistance. Here, the inciting or precipitating factors of abnormal states are emphasized.

SUMMARY.

1. The term "physiological inflammation" is suggested as a useful means to describe great variety of states, from a microscopical point of view, in which the resting or average nasal mucosae of human beings are found to occur.

2. The degree of physiological inflammation found is an index of the susceptibility of the patient to develop abnormal nasal states or disorders.

3. There are several distinct types of physiological inflammation characterized by definite relative changes in the amount or numbers of certain tissue elements. The clinical signs and symptoms exhibited by the patient can usually be surmised from these definite microscopical pictures.

4. The significant cells in the majority of tissue changes are the lymphocytes, plasma cells and eosinophiles. The eosinophiles are strikingly abundant in conditions that are commonly considered as purely vasomotor and anaphylactic, where there is no obvious neutroture of purulent exudation. In general, polymorphonuclear neutrophilic leucocytes seem to be of lesser importance than the above cells.

5. It is suggested that the development of an increasing state of physiological inflammation in the nasal mucous membrane is an expression of the lowering of resistance (in the wide sense used by immunologists) in that organ.

6. Abnormal nasal states, such as congestions with sneezing and watery discharge, polyps, polypoid and cystic formations, can apparently occur independently of any noticeable bacterial influence. It seems that in many people the proper functioning and resistance of the nasal mucosa is altered by reflexes sent to it from some other organ in the body which is abnormally stimulated; for example, from a skin which is sensitive to or has a lowered threshold to changes in temperature.

7. Bacteria are placed in a secondary role in the causation of abnormal states. A "preparing of the soil" seems to be necessary for them, just as there must be for proteins or chemicals, before they can become pathogenetically active.

FURTHER OBSERVATIONS ON AGRANULOCYTIC ANGINA.*

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Since the writer's first article has gone to press another case of exactly the same type has come under observation. It is even more remarkable, in that she was a sister of the first case reported.

Briefly, a review of the case is given. She was an adult female, age 42 years, single. The illness began on April 21, 1927, with a sore throat and a chill. Increasing prostration followed with diffuse submaxillary infiltration. The throat presented a reddening and edema. A few circumscribed areas of erythema appeared on the face and extremities. The temperature steadily mounted and death occurred in three days, April 27, 1927.

A smear from the throat showed numerous Gram-positive and Gram-negative cocci and a Gram-negative bacillus encapsulated, interpreted as Friedlander's bacillus. There were no diphtheria or Vincent's organisms. A white blood count was estimated at about 600. The cells on a smear were so scanty that a differential count could not be made. A blood culture gave a *pure growth of Friedlander's pneumobacillus.* This organism was definitely and positively identified by fermentation tests on the various sugars.

The positive blood culture confirms the author's previously expressed opinion that most of these cases are real septicemias. The low blood count is believed to be merely indicative of an overwhelming infection. She was a robust woman who had previously always enjoyed good health and certainly not the debilitated individual from whom a very low resistance would have been expected.

At first thought, heredity may seem to be a factor. However, on careful inquiry it is found that a sister-in-law, in no way a blood relative, died a few months ago in Charlotte with identically the same clinical manifestations. It is of interest to note that the latter patient was of the same family group.

These cases merit further study. Therapeutically, the situation so far has been hopeless. Fortunately, such infections are rare.

Davis Hospital.

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*From the Department of Head Specialties, Davis Hospital.

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FACIAL PARALYSIS ACCOMPANYING ACUTE MASTOIDITIS IN A DIABETIC PATIENT. RECOVERY OF FACIAL PALSY AFTER OPERATION.

Dr. Adolph Greenstein, New York City.

The development of a facial paralysis during the course of an acute otitis media and mastoiditis must always be considered a complication of great importance. It should be regarded as a suspicious symptom of bone destruction. Although the nerve often offers great resistance to destructive inflammations along its course, and may occasionally even resist inflammations after its bony covering has been destroyed, yet the presence of a facial paralysis does not always point to extensive bone destruction, but may occur coincident with an acute mastoid involvement, due to exposure.

When the paralysis is due to bone destruction, it need not necessarily be extensive, but may be, as pointed out by Von Trolch, a slight lesion extending in an unfortunate direction.

Facial paralysis may also be caused by the inflammation extending to the Fallopian canal, involving the sheath of the nerve without caries of its osseous canal. These cases prove the most favorable, for after the inflammation in the canal has subsided and the exudate absorbed, the paralysis completely disappears.

Whether a dehiscence in the Fallopian canal, as shown by Tomka, favors the development of facial paralysis has not been proven, for observation of Politzer and others show that the facial nerve lying free in the tympanic cavity, completely surrounded by pus, causes no symptoms of facial paralysis.

The seventh nerve may also be affected in the internal auditory meatus by extension of mastoid inflammation within the cranium, causing a meningitis or brain abscess; or by extension of the inflammation anywhere between the apex of the pyramid and the stylomastoid foramen.

The degree of facial paralysis varies, depending on whether the transmission is interrupted in certain bundles or in the whole nerve trunk. In the former cases, the different branches of the facial nerve are affected unequally. One, therefore, finds that sometimes the paralysis is more pronounced in the upper branches; that is, those

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extending towards the forehead and orbicularis palperbrarum, and at other times, in the lower branches, those extending towards the nose and angle of the mouth. As a rule, however, the orbicularis palperbrarum is less affected than the muscles of the nose and angle of the mouth.

In some cases, severe pain in the ear and corresponding side of the face often precedes the development of a facial paralysis. In other cases not the least pain is experienced and the paralysis is ushered in by a twitching of the facial muscles (spasmodic tic), or the paralysis may come on suddenly without any premonitory symptoms.

While the paralysis may be caused by a lesion of the nerve located centrally, during its course through the Fallopian canal or peripherally, it is desired in this paper to consider it only in its relation to acute mastoiditis.

In this connection, I desire to report a case of acute mastoiditis with facial paralysis of the same side.

The patient was admitted to the Oto-Rhino-Laryngological Department of the Beth David Hospital, on the service of Dr. Samuel J. Kopetzky, on March 4, 1923.

G. C., age 31 years, married; occupation, driver. *Habits:* Uses no alcohol, smokes cigarettes moderately. *Family History:* Negative. *Venereal History:* Denied.

Past History: Has never been ill up to three years ago, when he developed diabetes. Under diabetic treatment, he improved, and the glycosuria was reduced from 6 per cent to 3 per cent. He has never been operated upon.

Present History: Six weeks ago, at night, the patient suddenly awoke with a severe pain in the left ear. He consulted an otologist, who performed a myringotomy. The discharge continued for one week and stopped, followed by a great deal of pain. A second myringotomy was performed and drainage established, which was still present at the time of admission to the hospital.

Three weeks ago, he began to have pain and tenderness in the left mastoid; the pain being worse at night. A week later, he noticed that the left side of his face had lost its expression and appeared flat. He could not close his eyelids and the left corner of his mouth was dropped. He complained about some disturbance in the sense of taste.

Physical examination showed a patient fairly well nourished, with flattening of the left side of face, drooping of left side of mouth, and inability to close the left eye, wrinkle the forehead or whistle.

Pupils were equal, regular and reacted to light and accommodation. There was loss of sense of taste on anterior two-thirds of the tongue on the same side. Ear examination, presented a perforation in posterior inferior quadrant of left tympanic membrane, which appeared red and thickened. Posterior superior canal wall showed some sagging. Discharge was scant, thick and purulent, no swelling over mastoid region, although very tender over antrum and tip. Auricular fold was distinctly present.

HEARING TESTS.

Left Ear:	C fork 128	Air Cond., 10 Sec.	Bone Cond., 7 sec.
	C fork 1024	Air Cond., 15 Sec.	Bone Cond., 10 sec.
	C fork 2048	Air Cond., 0 Sec.	Bone Cond., 15 Sec.
Right Ear:	C fork 128	Air Cond., 20 Sec.	Bone Cond., 7 Sec.
	C fork 1024	Air Cond., 18 Sec.	Bone Cond., 14 Sec.
	C fork 2048	Air Cond., 20 Sec.	Bone Cond., 15 Sec.

Lateralized to the left side. Galton whistle heard. No spontaneous nystagmus, vertigo or past-pointing.

LABORATORY FINDINGS.

Smear from aural discharge showed pus cells and diplococci.

BLOOD COUNT.

White cells, 18,200. Polyn. leuc., 67 per cent. Blood sugar, .16.

URINALYSIS.

Sugar, 2.5 per cent. No acetone or diabetic acid. X-ray showed clouding of the left mastoid, with breaking down of bone trabeculae.

In view of the fact that this patient had diabetes, it was deemed advisable to treat him expectantly until he became sugar free.

On March 11 he developed a swelling over the lower part of his left mastoid, extending down to the posterior triangle of the neck. This swelling was very painful on pressure and the aural discharge also increased considerably.

On March 15 the mastoid operation was performed. On removing the cortex, there was a free flow of pus. Perforation took place through the digastric fossa into the posterior triangle of the neck to the extent of about 2 inches.

The lateral sinus was exposed as far as the emissary vein, which bled freely, appeared thickened and was covered with granulation. On curetting the diseased cells along the tegmen, the dura was exposed to the extent of about 1½ inches.

The bony covering over the aqueductus Fallopii appeared normal.

Drainage was established between the middle ear and mastoid cavity through the antrum and after all diseased cells were removed, the wound was packed. The tract to the posterior triangle of the

neck was also packed and caused to drain through the mastoid wound. The upper part of the wound was then sutured with two silkworm gut sutures and dressing was applied.

Immediately after the operation, the facial paralysis began to show signs of improvement and within six weeks there was almost complete recovery. The wound began to follow a normal course of healing, middle ear was dry within two weeks, and the mastoid wound was almost closed when, on the evening of April 27, he developed a most excruciating headache on the left side, and occiput, swelling of left cheek and neck, pain in throat, and inability to swallow. Temperature was 100° F. The headache was so severe that an opiate had to be administered.

At this time, he presented some rigidity of the neck, a positive bilateral Kernig, but no Babinski. He had perfect co-ordination and there was no spontaneous nystagmus, past-pointing or vomiting. The pulse slowed down to 60.

LABORATORY FINDINGS AT THIS TIME.

White blood count, 41,000. Polyn. leuc., 70 per cent. Blood culture, negative. Blood sugar, .145.

URINALYSIS.

Specific gravity, 1018. Sugar, .45 per cent. Otherwise negative.

SPINAL PUNCTURE.

Fluid clear, under tension. No increase in cells. Globulin, + —.

Due to the sudden change in the patient's condition, although the wound looked apparently well, it was deemed advisable to explore it in order to determine the cause.

On April 28 the old wound was reopened and granulations curetted. The entire sinus was laid bare and appeared in good condition. The dura exposure was then enlarged and found covered with a thickened fibrinous exudate. It appeared under some tension and it was thought best to explore the temporosphenoidal lobe. After the fan-shaped punctures in the usual directions were made, no pus was found.

The wound was then irrigated with saline, packed and a dressing applied.

On April 29 the swelling of the neck increased considerably in size and again extended to the posterior triangle of the neck. This time, a counter-incision was made at the lower end of the swelling to insure better drainage of abscess in the posterior triangle of the neck. The patient began to show immediate improvement and within six weeks made an uneventful recovery.

In order to determine the exact cause of the facial paralysis in this case, it would be necessary to exclude other conditions causing facial palsy.

First and foremost, it should be differentiated from a central lesion, in which the loss of power is never as complete as it is in peripheral lesion. In central paralysis, the eye on the affected side can usually be closed and the forehead wrinkled, whereas in peripheral they cannot.

Having excluded the above affection, there remain only two more possible conditions, *viz:* the ordinary garden variety of facial paralysis known as Bell's palsy and the condition due to extensive inflammation or necrosis of the mastoid cells. As in this case, the paralysis having improved following the mastoid operation, it would rather seem to favor a facial paralysis, secondary to mastoid necrosis, especially so when one considers the extensive destruction of bone tissue in diabetes.

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800 West End Avenue.

AN EPIDEMIC OF ACUTE OTITIS MEDIA CHARACTERIZED BY POSITIVE GASSEOUS PRESSURE WITHIN THE TYMPANUM.

DR. V. V. WOOD, St. Louis.

During the winter of 1925-26 several cases of acute otitis media were observed in which the predominant characteristic was a bulging drum membrane, evidently distended by positive pressure of air or some other gasseous substance within the tympanum.

The first cases observed were thought to be caused probably by blowing the nose in the presence of some "ball valve-like" action within the auditory tube. However, when several other cases had been seen and finally more than one in a family (three in one instance) appeared, the probability of it being something in the nature of an epidemic caused by some specific gas-forming organism was recognized. Cases continued to appear in the fall of 1926 and the following winter. Again in several instances more than one case would appear in the same family.

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The literature has not been carefully and completely searched, so that this may not be a new condition. However, the writer cannot recall having previously observed similar cases in such number as to suspect an epidemic, although occasional cases of ballooned drumheads have been seen at all times of the year.

No specific organism has been isolated as yet. The usual pyogenic bacteria have been found. A streptococcus has been present in every case cultured. One case also showed an unidentified spore-forming organism, but in no instance has a gas-producing tendency been evident in culture.

No case has been hospitalized early so it could be thoroughly worked up from a bacteriologic standpoint immediately upon paracentesis or before secondary infection would be probable. They should be cultured on a greater variety of media perhaps than has been done by the writer.

The constant appearance of a streptococcus may be significant. The more the writer sees of the streptococcus, the more he feels that it is the mocking bird of bacteriology and apes the clinical characteristics of many other bacteria, as syphilis does of many other diseases. Streptococci have been known to produce gas to some extent. However, the production of gas is an unusual characteristic of any definite type of streptococci known so far as the writer has been able to determine.

Appearance of the Drum on Otologic Examination. If seen early there may be only slight redness of the upper quadrants or perhaps a few enlarged and engorged vessels running towards the short process. The drum soon begins to bulge and frequently there is no redness or loss of transparency below Shrapnel's membrane. Occasionally the entire membrana tympani is so red and nontransparent that it is impossible to say what is the cause of bulging if seen for the first time late in the disease. Other drumheads show a surprising absence of redness and the gasseous cause of the distention is unquestionable. The hearing is always down.

Course of the Condition. In spite of the fact that a streptococcus has been found in every case cultured or examined bacteriologically, the condition has shown a surprising mildness of course and lack of the development of the usual much-feared streptococcic complications. There has not as yet been noted any especially virulent features or dangerous complications. No case of mastoiditis has yet appeared in the writer's experience. The temperature is not high and is usually about in proportion to the severity of the accompany-

ing upper respiratory infection which always precedes or accompanies the development of the otitis media.

One of four things usually happens:

1. Many cases subside spontaneously without going on to suppuration or the evident production of serous exudation.

2. Some cases become so markedly distended that paracentesis is necessary to relieve the intratympanic pressure and subsequent pain.

3. Other cases develop a serous exudate which is not apparently purulent.

4. A few have been seen to eventually develop a suppurative otitis media.

Thus it will be seen that the infection acts as does any other group of cases of acute otitis media, with no typical features except the positive gasseous intratympanic pressure.

In four or five cases the escape of gas or air upon paracentesis has been forceful enough to produce a distinct hiss, audible not only to the otologist but to the anesthetist, Dr. T. M. Davis. Needless to say, these cases were in great pain when incised.

There is rarely any difficulty in differentiating this condition from the so-called myringitis bullosa with blebs on the drum, which is so frequently seen accompanying influenza.

This is a preliminary report and the writer would be pleased to hear from other otologists as to whether they have observed similar cases and any additional remarks they care to make referrable to the condition.

To append a list of case reports would throw no more light upon the condition than the above brief description.

201 Humboldt Building.

CASE OF DIABETIS MELLITUS, ACUTE COALESCENT MASTOIDITIS LEFT, PERISINUS ABSCESS, a-SYMPTOMATIC, a-BACTERIAL, AND OBLITERATING THROMBOPHLEBITIS OF SIGMOID SINUS.*

Dr. Mark J. Gottlieb, New York City.

Mrs. A. M., age 67 years, admitted to the service of Dr. Samuel J. Kopetzky on Oct. 4 ,1926.

For three weeks before admission had pain in the left ear. Two days before admission left eardrum was incised by an ear specialist. Since then there has been a thick, purulent discharge from the left ear and patient has complained of marked weakness.

On admission there was slight tenderness over the left mastoid. Profuse purulent, thick discharge from the left ear. Glucose in the urine. Blood count: Hb., 67 per cent; R. B. C., 4,000,000; W. B. C., 11,800. Differential: Polys, 61 per cent. Mononuclears and transitionals, 39 per cent. Blood sugar, 190. Discharge from ear contained streptococcus mucosus capsulatus.

Eight days later the posterior half of the left drum seemed full, the discharge pulsated, and the quantity was increased. Two days after this, the Roentgenological department reported that there was marked absorption of the trabeculae and clouding of the left mastoid.

Oct. 15, 1926, simple mastoidectomy, left.

Findings: Coalescent mastoid containing thick pus in its entirety. Much granulation tissue present. Sinus exposed through its entire extent. Granulation tissue on sinus.

Procedure: Mastoid opened, pus evacuated, necrotic mastoid cells and granulations removed. Sinus found exposed from knee to bulb. Dura exposed surgically for about 1 c.m. Square in the floor of the middle cranial fossa. Wound packed with iodoform gauze and skin partially closed with three silkworm sutures.

Smears from the pus found in the mastoid showed streptococcus mucosus capsulatus.

The mastoid wound healed up to a certain point, and then no further progress was made. Wound edges had no granulations and looked anemic. The discharge was slight.

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From the Otolaryngological Service of the Beth Israel Hospital, New York City.
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Seventeen days after the operation patient complained of weakness and headache.

On Nov. 3 (twenty days after the operation), had a chill, temperature rose to 103.° Ten c.c. of clear spinal fluid under pressure was obtained on lumbar puncture. Patient did not hear with left ear (noise apparatus in right ear). There was a suggestion of Kernig and Babinski on the right side.

Nov. 5, neurological consultation. Increased tendon reflexes on the right side. Kernigs present on both sides. Babinski on the right side. Very little neck rigidity. Ankle clonus present on the right.

Impression: Brain abscess.

Leucocyte count on Nov. 3 was 14,000, with an increase in the per centage of polynuclear leucocytes to 67 per cent.

In view of the above, a revision of the mastoid wound was undertaken for the purpose of searching for additional disease and to decompress the cerebellum and temporosphenoidal region to prepare in this way this area for future surgical intervention into the cranium.

Nov. 4, revision of mastoidectomy. Gas and oxygen anesthesia. Findings: Granulations over sinus. Mastoid bone covering the middle cranial fossa very soft and necrotic.

Procedure: Granulations removed, sinus exposed far back, dura of middle cranial fossa and over the cerebellum exposed, wound packed with iodoform gauze packing and two silkworm sutures used to close skin.

Wound was dressed from time to time following the operation and the amount of discharge was slight. Wound edges did not have a tendency to produce healthy granulations.

Nov. 9: The dura was inspected at the time of dressing and the intracranial contents did not seem to be under pressure.

Nov. 10: Summary.

Chill and rise in temperature with concomittant rise in pulse nineteen days after operation. The temperature had continued low grade up to then. Her mentality is difficult to evaluate fully because of poor hearing. Patient is well oriented; responds fairly intelligently. Does not read or write. No disturbance in speech. No sensory disturbance. No astereognosis. Strength unimpaired, left side greater than right. Patient is left-handed. No headache, no dizziness.

Cranial nerves negative. No visual disturbances.

Eye grounds, some arteriosclerotic changes. Visual fields attempted, but could not be accurately mapped out, due to poor co-operation. Neck, no rigidity.

Reflexes, increased on right side. Babinski and confirmatories, right. Clonus, right. Bilateral Kernigs. No cerebellar disturbances. Today, patient has been somewhat drowsy.

Impression Brain abscess (temporosphenoidal).

Blood cultures taken on Nov. 6 and 8 show no growth.

Blood count: leucocytes, 16,500, with 87 per cent of polys.

Lumbar puncture: spinal fluid pressure was 18 m.m. of mercury. This was increased to 30 m.m. of mercury by pressure over the right jugular vein, and to 26 m.m. of mercury by pressure over the left jugular vein.

Three c.c. of cloudy fluid was obtained. The culture was negative and 10,000 cells per c.m., with 99 per cent of polynuclear leucocytes, was reported.

Nov. 11: It was decided to explore the sigmoid sinus and search for a collection of pus in the cerebellum and temporosphenoidal lobe. While the patient was being prepared for the operation, it was noted that she had a rigidity of the neck and stiffness of the extremities and gradually became comatose. The operation was performed without anesthesia.

Obliteration of Sinus and Ligation of Jugular Vein, Exploration of Brain—No pus obtained from cerebellar punctures. Temporal lobe under tension. No collection of pus found. Cerebrospinal fluid cloudy under pressure. Jugular vein apparently normal. Intima of sigmoid sinus lusterless and rough.

Procedure: Sinus covered by unhealthy looking granulations, sinus wall incised, no bleeding, although no clot was found. Sinus exposed further back, blocked above and below and incised in its entire length. No clot found. Pressure relieved from above. No bleeding. Sinus exposed still further back. Bleeding was obtained No clot observed.

Internal jugular vein ligated with two ligatures below omohyoid muscle. No bleeding obtained from lower portion of sinus. Pieces of sinus wall removed for examination. Cultures made from sinus. Visceral layer of sinus wall incised and cerebellum exposed with small linear incision, and search made for purulent collection with wide needle. No pus obtained. Cerebellum did not appear to be under pressure. Portion of squamous bone removed, dura incised—crisscross incision; cerebrum bulged into wound, temporal lobe searched by three punctures for abscess, with no results. When the needle was inserted parallel to the floor of the middle cranial fossa for about the distance of 1 inch, there was an escape of spinal fluid under pressure, which most likely came from a dilated ventricle.

Needle not inserted in any instance further than 3 c.m. Wound packed with iodoform gauze.

Culture from sinus sterile.

Pathological Report: Section of lateral sinus wall stained for bacteria, as well as general histology.

Section is that of a wall of a vein; the endothelium of the intima is not very distinct. The internal elastic lamina from the elastic stain appears fragmented. The media is edematous and the adventitia is very indistinct and fuses with chronic inflammatory exudate. On the intima side we find a blood clot which is made up of fibrin and red blood cells. To one side of the section the blood clot is much older and undergoing fibrous changes. It shows the presence of a great number of mononuclear and plasma cells and some fibroblasts and formation of new blood vessels. Bacteria not observed.

Opinion: Venous thrombosis.

Patient died twelve hours after operation.

Nov. 12: Post-mortem wound inspection: No clot present in lateral sinus. Cerebellum and cerebrum explored with large calibre needle; no pus found.

Cerebrospinal fluid in lateral ventricle, cloudy and under tension.

Cultures taken from wound, sterile. Cerebrospinal fluid culture, staphylococcus.

On admission, temperature was 100.2,° pulse 112, respiration 36. The temperature, pulse and respiration continued about the same, never going any higher, with remissions to approximately normal, reaching on Oct. 12 a temperature of 98.6,° pulse 80 and respiration 24. The temperature rose from that level and remained irregularly higher, not reaching in any instance above 101.2, which was on Oct. 22, with pulse 100 and respiration 24. It then declined and resumed its former level, until Nov. 3, when it rose with a chill to temperature 103,° pulse 116, respiration 32 and varied between 99° and 101.4° until Nov. 9, when it rose to temperature 103,° pulse 100 and respiration 28. Just before death, temperature rose to 105.6° pulse 114 and respiration 32.

Blood Sedimentation Tests (Fähraens): Oct. 14, 35; Oct. 15, 51; Oct. 16, 55; Oct. 18, 45; Oct. 21, 41; Nov. 4, 44; Nov. 10, 71; Nov. 11, 78.

The normal sedimentation is 4. The figures given demonstrate rather eloquently the trend of the patient's condition.

23 West 73rd Street.

LABYRINTHINE FINDINGS IN TWO CASES OF POSTERIOR FOSSA TUMORS.*

DR. RALPH ALMOUR, New York City.

Case 1: A married woman, age 29 years, had been complaining of a nervous breakdown and weakness for four years. This followed immediately after her last pregnancy, and was initiated by a tinnitus in the right ear. The tinnitus gradually increased in severity and she noticed some reduction in hearing in the affected ear. Two years later, she noticed that at times the right side of the face felt numb and that this numbness had become permanent within the past five months. During the past year, she had attacks of vertigo on changing from the horizontal to the upright position; the vertigo was in no particular direction. She staggered when attempting to walk. There was no headache. The blood and spinal fluid Wassermann were negative.

Neurological examination revealed a cerebellar disturbance in gait and station. There was a bilateral exophthalmos. The pupils were normal. There was a spontaneous horizontal nystagmus on looking to the left, rapid component to the left; a wide horizontal nystagmus to the right on looking to the right; a vertical nystagmus on looking upward, and a rotary movement of the eyeball when held in the midline. There was a weakness of both external recti muscles, especially the right. The right corneal reflex was abolished and there were marked sensory disturbances in the distribution of the right trigeminus. There was a slight facial weakness on the right side. The Romberg was positive. There was an ataxia of the right upper extremity. No adiadochkinesis. Pelvic girdle reactions were normal. The deep reflexes on the right side were markedly exaggerated. The ocular fundi showed a marked papilledema on the right side and to a lesser extent on the left side.

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*Read before the New York Academy of Medicine, Section on Otology, Dec. 10, 1926.

From the Otolaryngolocal Departments of Beth Israel and New York Polyclinic Hospitals, New York City.

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The functional examination of the inner ear follows:

TUNING FORK TESTS.

		RIGHT	LEFT	NORMAL
Middle C	air	24	49	45
	bone	15	21	20
C 2048	air	33	41	35
C 64	air	Heard	Heard	Heard

Weber Test: Lateralizes to left ear.

VESTIBULAR TESTS.

To test horizontal semicircular canals.

	Turning to RIGHT	Turning to LEFT
NYSTAGMUS	29 sec. to Left, horizontal in midline.	14 sec. to right, horizontal in midline
VERTIGO	15 sec. to left, then confused dizziness.	45 sec. to right.
PASTPOINTING	Touched with both hands	Left hand normal to left Right hand touched.

Caloric Tests (Water at 68°)

Head 30° Forward

	RIGHT EAR	LEFT EAR
NYSTAGMUS	After 4 ½ min., no nystagmus.	After 45 sec. marked increase in spontaneous horizontal to right. NO ROTARY element.
VERTIGO	None	Marked
PASTPOINTING	Touched both hands	Left hand to left. Right hand touched.

Head 60° Backward

	RIGHT EAR	LEFT EAR
NYSTAGMUS	No change from spontaneous nystagmus.	Marked hyperactive horizontal to right.
PASTPOINTING	Touched both hands	Left hand to left. Right hand touched.

The salient findings on functional examination of the labyrinth were: *1.* Right acoustic nerve deafness. *2.* Following rotary stimulation of the horizontal canals; *A.* Turning to the right produced a nystagmus twice as long as that obtained after turning to the left. *B.* Faulty pastpointing, particularly with right hand. *3.* Following cold caloric stimulation; *A.* Absence of all responses from the right ear with head up and 60° back. *B.* Perverted nystagmus on douching left ear with head up. *C.* Normal character, but hyperactive horizontal nystagmus on placing head back 60.°

Diagnosis: Right cerebellopontine angle tumor.

Basis for making the diagnosis: 1. History of vertigo, unilateral tinnitus, onset after pregnancy, staggering gait, numbness of face. *2.* Physical findings: Spontaneous vertical and horizontal nystagmus, ataxia, choked discs, facial nerve involvement, trigeminal nerve involvement. *3.* Labyrinthine findings of acoustic nerve deafness, absent responses from right vestibular stimualtion, perverted response from left vertical canal.

This patient was operated upon by Dr. Cushing and an acoustic neurofibroma of the right side was found at operation. The patient has made a complete recovery.

Case 2: A girl, age 12½ years, suddenly became dizzy and had a staggering gait. Prior to the onset of these symptoms, she had been in good health since the age of 6 years. Between the ages of 2 and 6 years she had had many convulsions, which were always associated with obstinate constipation.

The onset of the vertigo and staggering gait was sudden. It appeared while attending school in the early part of December, 1924. At first she had no other symptoms, but within four weeks she began to have obstinate constipation, headache and vomiting. There were no ear symptoms or any other sensory disturbances. There were no convulsions.

In January, 1925, she consulted Dr. K. Winfield Ney, who at that time found a weakness of the left facial muscles, an ataxic gait, positive Romberg, with falling to the left, a slight ataxia of both upper extremities, more marked on the right side, and a right knee-jerk of the Gordon-Holmes type. On testing the pelvic girdle the patient could be easily pushed backward. There was a spontaneous rotary nystagmus to the right and a horizontal nystagmus to the left. There was a vertical nystagmus upward. There was no spontaneous pastpointing.

The functional examination of the inner ear follows:

TUNING FORK TESTS.

		RIGHT EAR	LEFT EAR	NORMAL
Middle C	air	50	40	45
	bone	17	20	20
C 2048	air	35	35	35
C 64	air	Heard	Heard	Heard

Weber Test: No lateralization.

VESTIBULAR TESTS.

To test horizontal semicircular canals.

	Turning to RIGHT	Turning to LEFT
NYSTAGMUS	No alteration of spontaneous nystagmus to left. Spontaneous rotary to right not overcome.	Changed spontaneous rotary to horizontal which lasted 10 sec. and then returned to spontaneous rotary.
VERTIGO	None	None
PASTPOINTING	Touched both hands	Once to left with right hand then touch. Left hand touched.

Caloric Tests.

Head 30° Forward

	RIGHT EAR (Water 68°)	LEFT EAR (Water 110°)
NYSTAGMUS	After 2 min. slight rotary to left with eyes in midline. On looking to left, strong spontaneous horizontal appears.	After 3 min. 20 sec., cannot alter spontaneous horizontal to left or stop rotary to right. NO REACTION.
VERTIGO	None	None
PASTPOINTING	To right with right hand. To right with left hand once, then touched.	Touched both hands.

Head 60° Backward

	RIGHT EAR	LEFT EAR
NYSTAGMUS	Spontaneous horizontal seems to be increased in intensity.	Same as spontaneous and not increased as for right ear.
PASTPOINTING	To right with right hand. Touched with left hand.	Touched both hands.

The important findings on functional examination of the labyrinth were: *1.* Normal hearing in both ears. *2.* Following rotary stimulation of the horizontal canals; *A.* No nystagmus on turning to right; markedly diminished nystagmus on turning to left. *B.* Absent vertigo. *C.* Faulty pastpointing, particularly with left hand. *3.* Following caloric stimulation; *A.* Absence of all reactions from left ear. *B.* Delayed response from douching right ear, both with head up and back. *C.* Absent vertigo, both sides. *D.* Faulty pastpointing, particularly with left hand.

Diagnosis: Tumor of left lobe of cerebellum.

Reasons for diagnosis: 1. Signs of cerebellar inco-ordination and evidences of asynergia, major and minor. *2.* Labyrinthine findings: nonfunctioning left vestibular apparatus and normal left cochlear responses; impaired function of right vestibular apparatus.

The patient was operated upon by Dr. K. Winfield Ney. A bilateral cerebellar exposure was made and the left lobe of the cerebellum was found swollen and bound down by adhesions to the base of the skull. On freeing these and pushing the lobe aside, a large osteoma was found on the posterior surface of the petrous portion of the temporal bone close to, but not involving the internal auditory meatus. The tumor was not removed, but the patient has made a complete symptomatic recovery from the decompression alone.

Comment: Both these tumors were situated within the cranial cavity but outside of the brain substance. The vestibular tests in both cases resulted in fairly similar responses in that there was a destruction of function on the side of lesion and an impairment of function on the opposite side. The differentiating feature was in the response to the hearing tests. In the case of the angle tumor, an impairment of hearing of the perceptive type existed on the side of the intracranial lesion. In the case of the tumor of the petrous bone exerting pressure on the cerebellum, the hearing was normal on the affected side.

I am indebted to Dr. Ney for permission to present a brief synopsis of the history, physical findings and operation of the second case here reported.

51 West 73rd Street.

THE RELATION OF PROGRESSIVE DEAFNESS TO ENVIRONMENT.*

DR. D. C. JARVIS, Barre, Vt.

In devoting years to a study of silica dust inhalation, which it has been my privilege to do, it is necessary to study environment, both occupational and home, in an effort to estimate its influence upon the production of the clinical picture resulting from silica dust inhalation. The clinical picture resulting from silica dust inhalation and that of progressive deafness are so similar in many respects that one wonders whether the underlying fundamentals operating in each instance are not alike. The lungs by means of the bronchi and trachea communicate with the external air and the middle ear by means of the Eustachian tube likewise communicates with the external air. Both then to a certain extent are susceptible to the same respiratory environment, and both to a certain extent are influenced by certain clinical conditions of the nose and accessory sinuses. The pathologic changes in each instance are slight in their beginnings, more or less generalized in their distribution and slow in their development, the element of time being a necessary factor for the development of each clinical picture. Between ten and twenty-five years are necessary before a worker in silica dust presents himself for an examination of his lungs, and a like period of time seems to be often necessary before an individual presents himself for an examination of his ears. After observing the clinical manifestations of silica dust inhalation for years it is only natural that the conclusions resulting from these observations be applied to the problems of progressive deafness as one turns to clinical contact with these cases. In approaching the subject of progressive deafness from the viewpoint of environment, I realize that it is possible to do so from three different points of view. The first is represented by the viewpoint of the medical man interested in statistics. The second by the viewpoint of the clinical worker, who is ever interested in increasing his clinical efficiency and prefers a paper dealing with reports of cases in order that these cases may be studied, compared with his own and a decision made as to whether the treatment outlined is to be tried. The third, which is the viewpoint to be considered at this time, is that of the research worker who is con-

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stantly striving to answer the ever present why. Why, he asks himself, in spite of the various hearing tests, the mechanical methods of treatment and operative procedures upon the upper respiratory tract, do individuals still continue to grow hard of hearing? Is it possible there is some new approach to the subject of progressive deafness which may result in a new set of conclusions and a new attitude towards the hearing tests, symptomatology and treatment?

GENERAL CONSIDERATIONS.

Turning then to environment, it is realized that each individual after entering this life must adapt himself to his environment, whether it be that of the city or the country. In this process of adaptation he often gathers to himself a number of ills, among which may be mentioned progressive deafness. Each environment has its air we breathe, its food we take, its work we do, and its micro-organisms. In a city of the smaller type, such as Barre, Vermont, one sees round about him individuals who after living years of their life are unable to adapt themselves to the air they breathe, the food they take, and the work they do. As a result of failure in this process of adaptation they develop at times various clinical conditions having an insidious onset and slow rate of progress. As a result of observing all this one wonders whether progressive deafness, with its insidious onset and slow rate of progress, is not another clinical manifestation of the inability of the individual to adapt himself to his environment.

THE INFECTION PHASE OF ENVIRONMENT.

For the purpose of study it seems possible to divide environment into a number of phases, such as respiratory, dietetic, occupational and infection. Of these various phases the one that seems to readily gain attention is the phase of infection. While thinking over the infection phase of environment in connection with the etiology of progressive deafness I realize that the deductions and conclusions generally made are based upon the supposition that the infections of childhood, such as the exanthematous diseases, especially measles and scarlet fever, and later those of adult life are followed in certain individuals by progressive deafness. In this respect a theory seems to be developed approaching that commonly accepted in pulmonary tuberculosis, namely, the theory of childhood infection and, later, adult reinfection or superinfection. If interest is continued in this theory, then one becomes keenly interested in the work of the laboratory, the bacteriology of the upper respiratory tract and the various products of the laboratory which are supposed to be of assistance in combating infection. Clinical interest seems to center around the invading micro-organisms rather than the host, and energy seems to

be directed towards discovering and eradicating foci of chronic infection with the hope that by so doing the underlying fundamental cause of the progressive deafness is being removed. In endeavoring to explain why all individuals experiencing the infections of childhood and later those of adult life do not develop progressive deafness, the terms immunity and resistance are used without these terms being exactly defined. As a result of nineteen years' study of the clinical manifestations of silica dust inhalation it has seemed possible to me to formulate some definite conclusions regarding the role of infection in producing disease of the lungs. Without at this time going into detail, it is sufficient for the purpose of this paper to state that immunity may be defined as the ability of the individual to maintain his body free from lymphoid tissue. This ability is dependent upon his ability to control the fats in his diet. Immunity is also a matter of anatomy, it being the ability to maintain a satisfactory lymphatic drainage of the lungs under all conditions of respiratory environment. Resistance may be defined as the ability of the individual to maintain a satisfactory alkali reserve, this ability depending upon his ability to control the acid producing and alkaline producing foods in his diet. The first requirement for the development of infection in silica dust inhalation is a lowering of the alkali reserve, the second an inefficient lymphatic drainage of the lungs, and the third the presence of an increased amount of lymphoid tissue. Let us apply these conclusions reached after nineteen years' study of silica dust inhalation to some of the problems surrounding progressive deafness and note the result.

FIRST CONCLUSION.

It has just been stated that the first requirement for the development of infection of the lungs in silica dust inhalation was a lowering of the alkali reserve. Perhaps a brief explanation is in order as to how this conclusion was reached. During a study of silica dust inhalation I observed that when olive oil was added to a weak solution of silicic acid in a test tube, an emulsion having the appearance of milk resulted. Previous to this time I had observed that when an individual was suffering the early ill effects of silica dust inhalation a subsidence in cough and expectoration and a gain in weight took place when oil, such as olive oil or mazola oil, was added to the diet. This improvement required from four to six weeks. Following the test tube observation, I assumed that inhaled silica dust with the passing of time in some manner liberated silicic acid within the body and the reason results had been secured from the use of oil was because in some manner there had probably been produced within the body an emulsion similar to that observed in the test tube. As a

result of these observations sodium bicarbonate was used when granite cutters presented themselves for relief of various respiratory conditions. By its use it was possible for the first time in eight years to prevent further development of many of the respiratory conditions these men presented. Lung abscess, the appearance of which had been almost inevitable in the clinical sequence each man presented, was for the first time prevented from making its appearance. With the same number of men working it has been possible by means of alkaline treatment to reduce the incidence of illness from a permanent chest condition from 45 to 22 per 1,000 men.

During the experimental use of sodium bicarbonate the upper respiratory tract was closely studied in an effort to determine whether some special appearance might serve as a basis for the administering or discontinuing of sodium bicarbonate. Silica dust as it is inhaled does not produce an increased redness of the mucous membrane lining the nose in a granite cutter, but the men coming up for clinical relief showed a marked increase in redness of the mucous membrane lining the nose, which did not seem to be dependent upon an acute condition of the upper respiratory tract or a local condition, such as a sinus involvement. This redness decreased under the administration of sodium bicarbonate. With the passing of time the degree of redness of the mucous membrane lining the nose came to serve as a basis for the giving or discontinuing of alkaline treatment. These results seemed to hold good when checked up on individuals not associated with the granite cutting industry. Turning to clinical contact with cases of progressive deafness, it was natural when a marked increase in redness of the mucous membrane lining the nose was encountered, which did not seem to be dependent upon an acute condition of the upper respiratory tract, or a local condition, such as a sinus involvement, that sodium bicarbonate be prescribed. As a result of its use I observed that the feeling of increased stuffiness in the ears which the patient complained of was often relieved in a short period of time, twenty-four hours many times being sufficient to bring about relief. About half of the patients volunteered the statement that tinnitus was either relieved or had disappeared. Hearing tests taken at subsequent office visits showed an increase in hearing for the 256 and 64 forks. At first it was thought the improved results were a coincidence. In order to determine if this was so, catheter inflations and local applications were discontinued, but the same relief continued. The only logical conclusion that seemed possible was that in some individuals a lowering of the alkali reserve manifests itself in the middle ear and Eustachian tube.

Utilizing this conclusion in attacking progressive deafness from the preventive side, patients suffering from progressive deafness have been encouraged by means of diet lists to select their food from a list of alkaline producing foods as far as was practical and to take sodium bicarbonate when they noticed a return of a feeling of increased stuffiness in the ears or the tinnitus. Patients who have co-operated by reporting from time to time state that their exacerbations of impaired hearing are less frequent and when they do appear are often controlled within forty-eight hours. Previous to the sodium bicarbonate observation I had observed that in patients suffering from progressive deafness a candy spree on Sunday, for example, resulted in a feeling of increased stuffiness in the nose and further impairment of hearing lasting until about the following Thursday or Friday. Since the sodium bicarbonate observation I have noted that if such an individual will take two doses of sodium bircarbonate before the anticipated candy spree and two doses afterwards, that most always the nose and ear symptoms will not appear.

SECOND CONCLUSION.

Another requirement for the development of infection in silica dust inhalation is the presence of an increased amount of lymphoid tissue in the respiratory tract. Perhaps a brief explanation is also in order as to how this conclusion was reached. In the early days of a study of silica dust inhalation it seemed advisable to determine, if possible, the type of individual lasting reasonably long in the granite cutting industry and the type breaking down early. In an effort to determine these two types, 500 granite cutters actually at work were examined, as well as those representing the survivors in the industry and those who had broken down early. As a result of this work it seemed possible to conclude that an individual was susceptible to the early ill effects of silica dust inhalation in proportion to the amount of lymphoid tissue present in the upper respiratory tract. While carrying on the Roentgen ray phase of the silica dust study, I discovered by means of serial films that certain circular densities appearing upon the chest Roentgenograms disappeared during absence from work and reappeared on re-entry into the industry. These densities when checked up with autopsied lungs proved to be densities of lymphoid tissue. Soon after this the pathologist, who was interested in experimental pathology, reported that guinea pigs subjected to silica dust inhalation showed an anatomic increase in lung lymphoid tissue as lung lymphatic drainage became less efficient. Following the above observation and report, I concluded that by means of the circular densities appearing on the chest Roentgenograms the behavior

of the lung lymphatic drainage was being observed. The question then arose as to whether a relation existed between the amount of lymphoid tissue in the upper respiratory tract and the number of circular densities appearing upon the chest Roentgenogram. In an effort to answer this question, I made a comparative study, with the result that it seemed possible to finally conclude that when there is an increased amount of lymphoid tissue in the upper respiratory tract, as evidenced by increased size of turbinates, especially lymphoid nodules upon the posterior pharyngeal wall and tonsils larger in size than you would expect to find them, there is also an increased number of circular densities on the chest Roentgenograms.

This observation was considered of sufficient importance to warrant a control study. For this purpose Roentgenograms were made of the chests of 105 students of a college preparatory school and a detailed study was made of their upper respiratory tract. In order that pulmonary disease might be ruled out, two medical men who devoted all their time to the diagnosis and treatment of pulmonary disease were brought to Barre for the examination of the chests of these students. As a result of this control study it seemed possible to still maintain the original conclusion that when we had an increased amount of lymphoid tissue in the upper respiratory tract we also had an increased amount of lymphoid tissue in other parts of the respiratory tract, as shown by the increased number of circular densities appearing on the chest Roentgenograms. Having observed that certain individuals had more lymphoid tissue in the upper respiratory tract than others, the question arose as to why this was so. In an effort to answer this question a great deal of work was done, it seeming possible to finally conclude that lymphoid tissue was present in the upper respiratory tract in proportion as fats were absent from the diet. Following this observation when an examination of the upper respiratory tract showed the presence of an increased amount of lymphoid tissue, cod liver oil was prescribed as a means of adding fats to the diet. Feeling this increased amount of lymphoid tissue was evidence of an inefficient lung lymphatic drainage, hot applications were applied to the chest externally when a clinical condition developed, with a hope that by their means the lung lymphatics would be dilated and lung lymphatic flow be thus hastened. In addition men were encouraged to wear respirators in order that silica dust be kept out of the lungs.

Turning to clinical contact with cases of progressive deafness from force of habit I continued to estimate the amount of lymphoid tissue present in the upper respiratory tract, feeling as I did so that I was

at the same time estimating the amount of lymphoid tissue present in the membranous portion of the Eustachian tube. That this tissue represented suitable soil for the growth of micro-organisms and in proportion as it was present in like proportion might one expect from time to time exacerbations of the aural condition. Also in proportion as lymphoid tissue was present in like proportion, might one expect an inefficiency of the lymphatic drainage of the middle ear, and in proportion as the lymphatic drainage of the middle ear was inefficient, in like proportion might one expect that chain of pathologic conditions which follow in the wake of an inefficient lymphatic drainage system, fibrosis being the last member of this chain. It was only natural, following the silica dust experience, that cod liver oil be prescribed when there appeared to be an increased amount of lymphoid tissue in the upper respiratory tract; that warm oils be used in the external auditory canal night and morning in an effort to dilate the lymphatics of the middle ear and thus hasten lymphatic flow. Oil suitable for use in the nose was dropped in each side of the nose night and morning in order that dust particles 10 microns or under in size in the inspired air might be caught on the oily surface and the lymphatic drainage of the middle ear be spared by making it less possible for particles of dust of microscopic size to gain entrance to the middle ear through the Eustachian tube. Following the use of these measures I observed that patients suffering from progressive deafness showed improvement in hearing for the voice, whisper and acoumeter. Why improvement for intensity as manifested by these three tests should take place and not improvement in hearing for the forks I do not know.

PLACE ENVIRONMENT OCCUPIES IN THE TREATMENT OF PROGRESSIVE DEAFNESS.

From the preceding observations and deductions, the only conclusion that seems possible is that individuals complaining of progressive deafness are more susceptible to their environment than others. That this susceptibility is manifested in some by an increase in the amount of lymphoid tissue present in the upper respiratory tract. This tissue is present in proportion as fats are absent from the diet. When this state of affairs exists there appears in these individuals to be a diminution in hearing for the voice, whisper and acoumeter. The use of warm oils in the external auditory canal for the reasons already given, the use of oil in the nose and the taking of cod liver oil by the patient seem to bring about an improvement in hearing for the above tests. In other individuals susceptibility to environment is manifested by a lowering of the alkali reserve as manifested in an

increased redness of the mucous membrane lining the nose. When this condition exists there appears to be a diminution in hearing for the 256 and 64 forks. The use of sodium bicarbonate and alkaline producing foods as a means of increasing the alkali reserve seems to increase hearing for the tuning forks mentioned. If one cares to look upon progressive deafness as being a local manifestation of the inability of the individual to adapt himself to his environment, then having demonstrated that a case of progressive deafness exists, the first step seems to be to study more closely the upper respiratory tract in an effort to estimate the amount of lymphoid tissue present in order that a decision may be made as to whether fats shall be added to the diet or cod liver oil used. The second step seems to be to study carefully the degree of redness of the mucous membrane lining the nose. On the results of this study rests the decision as to whether alkaline treatment is to be used. For the one who is primarily interested in clinical medicine, what would a clinical paper prepared from the viewpoint of environment tend to show? It would endeavor to prove by report of cases that a loss of hearing for the 256 and 64 forks suggests a lowered alkali reserve. That a loss of hearing for voice, whisper and acoumeter suggests the need of vitamine A. The progress of the cases would endeavor to prove that the use of vitamine A or the raising of the alkali reserve brought about improvement in hearing for the tests mentioned above. To consider and enlarge upon these clinical phases would unduly prolong this paper. In the present paper I have tried to apply two conclusions reached after years of study of silica dust inhalation to some of the problems surrounding progressive deafness, feeling that each one reading this will, if interested, work out his own cases.

SUMMARY.

1. For the purpose of study it seems possible to divide environment into a number of phases, such as respiratory, dietetic, occupational and infection.

2. A consideration of the infection phase of environment tends to lead one to conclude that while a relationship may exist between infection and the onset of progressive deafness, this relationship apparently seems to be a secondary one, rather than a primary one.

3. A consideration of the dietetic phase of environment tends to lead one to conclude that lymphoid tissue is present in proportion as fats are absent from the diet.

4. Lymphoid tissue apparently repre ents suitable soil for the growth of micro-organisms. When there is an increased amount present in progressive deafness there seems to be a diminution in

hearing for intensity. The proof of this seems to be the increase in hearing for intensity when fats, such as cod liver oil, are added to the diet.

5. A lowered alkali reserve apparently represents suitable preparation of the soil. This alkali reserve is estimated by a study of the degree of redness of the mucous membrane lining the nose. When there is a lowering of the alkali reserve in progressive deafness there seems to be a diminution in hearing for pitch. The proof of this seems to be the increase in hearing for pitch when alkaline treatment is used.

6. A consideration of the various phases of environment tends to lead one to conclude that progressive deafness may possibly be a local manifestation of a nutritional disaster.

7 Quarry Bank Building.

LIGHT THERAPY: ITS INDICATIONS AND METHOD OF APPLICATION IN OTOLARYNGOLOGY.*

DR. MAURICE WEISBLUM, Philadelphia.

A thorough knowledge of the physical properties and physiological effects produced by the various rays upon living tissues is of the utmost importance for the proper employment of radiant energy in the treatment and amelioration of pathological conditions.

It is therefore necessary to describe the different rays before proceeding with the discourse on their therapeutic value.

The following rays are employed as therapeutic agents at the present time: The solar, red, infrared, blue, ultraviolet, X-ray and radium rays.

The solar rays, coming from the sun and constituting a combination of all enumerated above, with the exception of the X-ray and radium emanations, have both heat producing and actinic or chemical properties. The solar rays, as we all know, have been utilized from time immemorial, with beneficial results in the treatment of constitutional and local diseases.

But the sun does not shine every day and so we cannot obtain solar rays when it is cloudy or raining. Then, many of the ultra-

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violet rays, especially those of the very short wave lengths, are absorbed by the particles of dust in the atmosphere. This explains why the sun's rays are more beneficial at high altitudes and at the seashore. At the latter place the individual also receives the benefit of the rays reflected from the sand and thus receives a double dose. Ordinary glass absorbs the ultraviolet rays whose wave length is less than 3,200 Angstrom units, which explains why people do not tan sitting in glass-enclosed rooms. There is now, however, manufactured a special glass which does transmit rays whose wave length is less than 3,200 Angstrom units, thus overcoming that difficulty. Wave length is the distance between two crests or two troughs of the wave, or vibration in the ether, and is the speed of light divided by the number of vibrations per second. An Angstrom unit is one-ten millionths of a millimeter.

The infrared is a ray whose value as a therapeutic agent has only been recognized in recent years, and which requires much study and research. This ray occurs at the near end of the spectrum and has a wave length of more than 7,700 Angstrom units. It is an invisible ray, because the vibrations are too slow to form an impression upon the retina.

The infrared ray is produced by glowing carbon arcs, an alloy of carbon and iron, and other metal electrodes. And also by very hot black bodies. The lamp which predominates today is one containing carbon electrodes, and consuming as much as 1,500 Watt hours. One-hour exposures will accomplish the desired result. When weaker lamps are used the duration of treatment must necessarily be increased.

This ray penetrates the tissues to a depth of about 10 inches, and when absorbed, heat is generated.

The strength of the rays is directly proportional to the heat and size of the electrodes and inversely as the square of the distance.

These rays, due to their deep penetrability and heat production, induce a deep hyperemia, an increased circulation in the part exposed, and a consequent absorption of the inflammatory exudate. Since there is a considerable rise in temperature, it is believed that many of the bacteria are thus destroyed. The rays also have a sedative effect upon the nerves. These rays are utilized chiefly on account of their heat production in the deep tissues, and are therefore of benefit in the early stages of mastoid disease; but are contraindicated when pus is present.

In trifacial neuralgia they exert a soothing influence and at times relieve the pain completely.

The infrared rays help to relieve the congestion in otitis media, and also to ameliorate the pain.

They will hasten suppuration in furuncles of the ear and nose, and may also abort them if applied at the onset.

The exposures to the infrared ray should last at least one hour daily at a distance of about 3 feet. In severe cases, two treatments daily may be necessary.

The ultraviolet rays are derived from heated carbon electrodes or from the mercury quartz lamp perfected by Cooper-Hewitt, and later by Kromayer.

About thirty years ago Funsen perfected a carbon lamp which generated rays equivalent to the solar but rich in ultraviolet rays of short wave lengths, less than 3,200 Angstrom units.

Later, Cooper-Hewitt demonstrated that a current passing through vaporized mercury enclosed in quartz generated almost pure ultraviolet rays, some of which are as low as 2,000 Angstrom units.

This is an air-cooled lamp.

Kromayer perfected a water-cooled mercury quartz lamp which enables one to employ quartz attachments, by which cavities can be exposed to the rays. These rays are of short wave length, and are invisible because their vibrations are too rapid to produce an impression on the retina.

The ultraviolet rays are actinic or chemical in character and produce chemical changes in all matter upon which they impinge. They penetrate to a depth of about one-thirty-second of an inch and when the tissues are compressed the penetration is two or three times as great. They produce tanning of the skin by the deposit of pigment from the blood in the capillaries. Some observers believe that this radiant energy is absorbed by the blood and that the endocrine glands are stimulated thereby, and that the calcium and phosphorus content of the blood is raised.

It is important to know that individuals react differently to exposure to the ultraviolet rays, and that blondes will react more strongly than the brunettes. When treating superficial lesions it is advisable to remove all scabs and crusts, otherwise the rays will not reach the diseased parts.

The ultraviolet rays destroy the tubercle bacilli very rapidly, so that they are an important adjunct in the treatment of tuberculous ulcers of the tongue, pharynx and larynx. With suitable quartz applicators the rays can be directed to the lesions.

In chronic rhinitis the rays exert a stimulating influence upon the mucosa and promote a restoration of normal function. The writer

has obtained favorable results in patients with sinus disease, relieving the headache and sense of fullness. It is difficult to explain the modus operandi in these cases, as well as those with bronchial asthma, yet some of the patients are relieved just the same.

It is my belief that the large quantity of ozone generated while the lamp is burning exerts a beneficial influence.

In wounds resulting from mastoid operations where the healing had been protracted, we were able to stimulate the tissues and to hasten the closing of the wound.

In treating external lesions it is advisable to give four-minute exposures at a distance of 30 inches at intervals of three or four days, depending upon the amount of erythema produced.

Mucous membrane and cavities may be exposed for twice the length of time and at more frequent intervals.

As the treatments continue the time of exposure is increased and the distance is decreased.

The eyes of the operator, nurse and patient should be protected with colored glasses to prevent conjunctivitis.

I have seen severe cases of conjunctivitis in operators who were careless, but these cleared up promptly when proper care was observed.

There is only one instance in the field of otolaryngology where the utraviolet rays must not be used, and that is when malignancy is present. These neoplasms are stimulated and grow very rapidly after exposure to the rays.

The blue and red rays are only useful because of their heat production but have no power of penetrating tissue.

References: Recent Progress in Phototherapy and Apparatus. Frank T. Woodbury.

CONCLUSIONS.

1. Physicians should possess a thorough knowledge of the physics and physiological action of radiant energy in order to properly apply this modality.

2. Light therapy is a useful adjunct in the treatment of disease, but is not a panacea for all ills.

3. Light therapy is of material aid to the otolaryngologist if applied intelligently and when indicated.

1638 S. Broad Street.

REVERSE TRACHEOTOMY* (AN ORIGINAL METHOD FOR RAPID TRACHEOTOMY, WITH A NEW INSTRUMENT).**§ PRELIMINARY REPORT.

Dr. M. Joseph Mandelbaum, New York City.

The antiquity of tracheotomy has long been established. The development of the modern operation with its various refinements in technic has produced very few essential changes. The entry from "without" into the tracheal lumen, and the insertion of the tracheotomic cannula, is basically the same procedure as originally performed.

History: In order to establish the priority of a "reverse" method of tracheotomy, I made a careful study of the history of the operation in Wright's[1] excellent work. It revealed the interesting information that "although Sprengel asserts (VII, 144) that Frederic Dekkers was the first to recommend 'paracentesis' of the trachea, Sanné in a work on bronchotomy, published in 1694, states that, according to Malavicini, Sanctorius, who died in 1636, first made use of a trocar, the cannula of which he left in the wound for three days."

This procedure, called "laryngocentesis", was also described in 1784 by Garengot[2].

It appears, therefore, that Sanctorius, nearly three hundred years ago, utilized an instrument constructed on the principle of my reverse tracheotome to enter the trachea from *"without"*.

The idea, however, of utilizing a similar instrument to make a tracheotomic opening from "within outward" originated with me in an emergency, without knowing of the previous use of a similar instrument, for the same purpose but in a *"different"* manner.

The origin of the modern tracheotomy tube is attributed by Holmes to A. G. Richter[3], who published his Obs. Chirurg. in 1776.

I believe, therefore, at least as far as a careful study of the history and literature reveals, my method of reverse tracheotomy to be an entirely original one.

Tracheotomy with Use of Intratracheal Guide: The operation for tracheotomy, especially in unskilled or untrained hands, is always a difficult procedure. Indeed, even the experienced, at times, meet

*From the Bronchoscopic Clinic and Department of Experimental Surgery, Hospital for Joint Diseases.
**Read with moving picture demonstration before the Harlem Medical Association, New York, Jan. 5, 1927.
§Demonstrated before the Section on Laryngology, Rhinology and Otolaryngology, Washington, D. C., May 16-20, 1927.

with cases that try one's skill and resourcefulness. The operation of election may be a simple enough procedure, but an emergency tracheotomy with a strangling, struggling individual, offers a proposition that may present the apparently impossible. No time may be lost and immediate action is needed if a life is to be saved. Even in tranquil patients, the pretracheal tissues may be swollen with a suffusion of blood and the classic anatomic landmarks, ordinarily found with ease, are difficult if not impossible to find.

Under these conditions at least, an easier, more certain and more rapid technic is welcome. So distressing at times has been the difficulty in performing this operation that various means have been devised to simplify it. More recently attempts have been made by Lewis[4] to use a bronchoscope as an intratracheal guide as a preliminary step to cutting down upon the trachea. This method is of particular value with transillumination of the trachea by means of the lighted bronchoscope, in cases of collapse of the trachea due to pressure on its lumen from without, and upon reading his article one can readily appreciate, in certain cases at least, the value of a "reverse" method of tracheotomy. A similar method has also been described by Gill[5].

It is not the intent of the writer in this article to discuss the various indications for this method of performing tracheotomy, nor to weigh the comparative merits of the classic, orthodox procedure and those of "reverse" tracheotomy. I will here rather describe the instances leading up to this operation and a description of same in both the animal and human subject.

Emergency Operation: On May 14, 1924, while making a social visit upon a medical colleague, he received an emergency call to a child residing in the same apartment house. The messenger said the child was choking. Being asked to accompany him, we hastened to respond and found the patient to be a robust male child, age 5 years, who one could see at a glance to be in imminent danger of asphyxiation. His face was swollen and livid, the pupils widely dilated, the supraclavicular fossae deeply retracted, and he seemed to be suffering from a tonic laryngospasm. Bloody, frothy mucus was dribbling from his mouth. The father said the child had recovered about ten days previously from severe measles, with croup.

Being wholly unprepared for such an emergency, I succeeded, by forcibly opening the patient's mouth with the aid of a wooden tongue depressor, in pulling the tongue forward; whereupon the child took a few short inspiratory gasps. Each time, however, upon relaxing my hold of the tongue, respiration would cease immediately. Instructing

my colleague to continue the above-mentioned procedure, I hurried to his office below, hoping to find a suitable instrument for an emergency tracheotomy.

Rapidly glancing over the contents of his instrument case, I noticed an old curved Wylie vesical trocar and cannula (Fig. 1). As I was about to return to the patient's apartment, I was notified that he had again ceased breathing. Hastening back, I found him pallid,

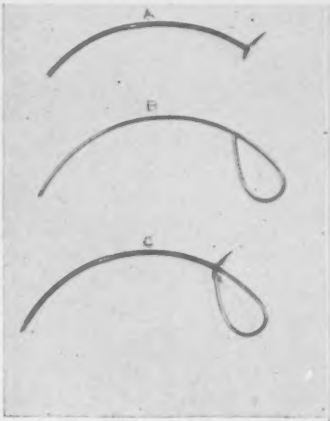

Fig. 1.

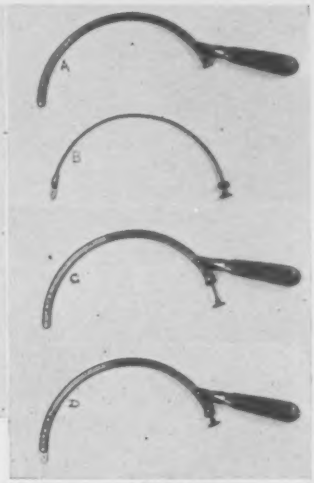

Fig. 2.

Fig. 1. The Wylie vesical trocar and cannula used for original emergency reverse tracheotomy. (a) Cannula and (b) trocar dissembled; (c) trocar and cannula assembled.

Fig. 2. "Reverse Tracheotome" used for animal and human experimental work. (a) Tracheotome cannula with handle. (b) Knife-bearing shaft with knife blade at distal end and "push-button" extremity at proximal end. (c) Cannula and knife-bearing shaft assembled, "push-button" (proximal) extremity "withdrawn" from proximal (handle) end of cannula, causing distal knife end to be pulled back into and concealed within distal end of tracheotome cannula. (d) Same as (c), but with push-button end of knife shaft pushed "home", and knife end of curved shaft emerging from distal end of tracheotome cannula.

limp and apparently dead. I immediately forcibly opened his mouth, inserted the vesical cannula with trocar inside of it, forced it between the patient's vocal cords and down into the trachea and, fixing the epiglottis with the index finger of my left hand, much in the manner as one does to introduce an intubation tube by the O'Dwyer method, pushed the trocar point through the midline of anterior wall of the trachea and the neck tissues in front of it, from *"within outward"*, meanwhile instructing those assisting me to continue artificial respiration.

The trocar was quickly withdrawn and the cannula, acting at the same time as a combined intubation and tracheotomy tube, caused a deep inspiratory rush of air into the lungs. The child, aided by the artificial respiration, began after a few exciting and stormy moments to breathe noisily, though regularly. The pallidness soon disappeared.

A messenger in the meantime had been dispatched for instruments, and a tracheotomy tube was introduced through the wound to replace the vesical cannula. This was done about forty minutes after the emergency tracheotomy had been performed. After three weeks' battle with a severe streptococcus laryngotracheitis, the cause of his attack, the child made a complete recovery.

Experimentation: This experience, which I reported at the May, 1925, meeting of the American Bronchoscopic Society[6], coupled with

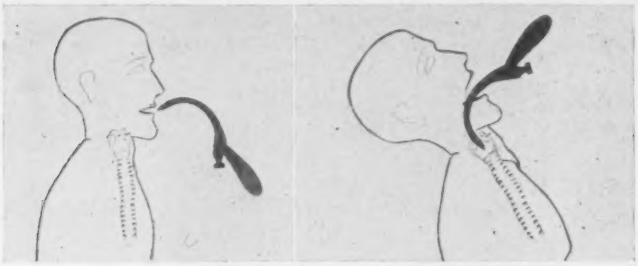

Fig. 3. Fig. 4.

Fig. 3. Position of Reverse Tracheotome about to be introduced into mouth (diagrammatic).
Fig. 4. Instrument introduced into mouth and just entering larynx between the vocal cords. Note beginning extension of head on neck to accommodate passages to shape of instrument.

further research I was conducting upon the trachea and bronchi of lambs, rabbits and fowls, which was published nearly two years ago[7], impressed me with the possibilities of devising a special instrument for performing a "reverse" tracheotomy.

The instrument I am about to describe and which I call the "reverse-tracheotome (Fig. 2) is the result of considerable experimentation on animals, cadavers and patients, the latter previously tracheotomized by the direct orthodox method for various pathologic conditions.

Thus having accidently determined the mechanical possibility of accomplishing a tracheotomy from within outwards by means of a curved, sharp pointed instrument, introduced into the open mouth, down between the vocal cords and into the tracheal canal, I set about

to find out what form of arc was described in the passage of such a curved instrument through the above-named channels.

By means of a curved flexible wire passed into the mouth, throat, larynx and trachea of a cadaver, with the head in the "anatomic" position, the various angles through which a curved instrument must pass, and the arc described in its passage, was ascertained. An attempt was then made to pass the wire bent in the form of the arc above described, with the head of the cadaver *"flexed"* forwards on its chest, in various degrees, from a slight to a complete flexion.

It was noted that the arc described by the bent wire in its travel along the above-mentioned passages with the cadaveric head in the *anatomic* position, could with but slight deviation, be easily passed

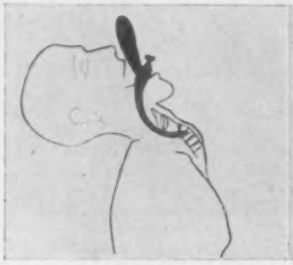

Fig. 5.

Fig. 6.

Fig. 5. Distal end of instrument in the tracheal canal, against its anterior wall between second and third tracheal rings. Note anterior bowing of trachea and corresponding bulging in anterior midline of neck. Compare with Fig. 7. Note (see Figs. 6 and 8) that as soon as tracheotome pierces skin, the bulging disappears and trachea assumes its normal position.

Fig. 6. Knife end of tracheotome penetrating midline of anterior tracheal wall and pretracheal tissues, and cannula emerging from reversely made tracheotomic wound.

through these channels with the head held in various degrees of flexion.

Then with the cadaveric head placed in various degrees of *"extension"*, from slight to complete extension, with but slight change in the curve of the wire, it was found that it could be just as readily passed along these channels as in the *anatomic* and *flexed* positions. Thus I determined the "mean" arc of the passages through which such an instrument must pass and this was the pattern upon which the curved cannula of the reverse tracheotome was made. This arc I have termed the "orotracheal arc".

Description of Reverse Tracheotome: The instrument used in my animal experimentations and which you will later see in the motion picture demonstration, consists of a somewhat scythe-shaped, flat-

tened, hollow cannula curved on its long axis in an arc ranging about one-half of a circle. A cross-section of the cannula resembles a somewhat flattened oval. Near the upper (proximal) end of the cannula, attached at an angle of 20° from its convex (superior) border is a handle, beneath the attachment of which the cannula terminates widely opened, and upon the threaded extremity of which a rounded screw cap is placed. Through the center of the screw cap is a small oblong slot, through which passes a curved steel shaft terminating in a knife-like end. At its lower (distal) extremity the cannula terminates with its end smoothly rounded off and with a vertical "slot-like" orifice, through which the knife end of the shaft can be passed back and forth. The terminal (distal) portion of the cannula is staggered on

Fig. 7.

Fig. 8.

Fig. 7. Showing Reverse Tracheotome in position; its blunt distal end pushed against midline of anterior wall of trachea, causing visible forward bulging of pretracheal tissues; operator's right index finger palpating distal blunt end of instrument, over which by means of a slight upwards and downwards motion of its handle, the first to fourth tracheal rings can be felt to "roll", previous to selecting point to be pierced by concealed knife. Compare with Fig. 5.

Fig. 8. Knife end of tracheotome penetrating midline of anterior tracheal wall and pretracheal tissues, and cannula emerging from reversely made tracheotomic wound.

its upper convex and lower concave borders as well as upon both its sides; these small perforations serving as additional breathing holes.

Running through the entire length of the flattened arc-shaped cannula is a stiff steel shaft, conforming to the shape of the cannula, terminating at its distal extremity in a sharp bistoury-shaped knife blade, and at its upper (proximal) end with a button-like thumb piece, pressure against which serves to force the knife end against and through the mid-line of the anterior wall of the trachea and pretracheal tissues.

A rather stiff coiled spring at the upper end of the cannula, pressing against the under surface of the button-like thumb piece, serves to keep the knife end of the curved steel inner shaft, hidden inside of the terminal opening of the cannula.

Technic of Operation: In emergencies the instrument can and may needs be used without preliminary local anesthesia. In operations of election, as in the first step of a two-stage laryngectomy, both intratracheal cocain anesthesia and subcutaneous novocain infiltration of the pretracheal tissues is applied, after a proper preliminary asepsis of the orotracheal mucosa by the thorough application of a 1 to 250 acriviolet solution to the entire mucous surface of the mouth, pharynx and larynx. The patient may be held in a sitting or horizontal posture. The operator faces the patient, holding the handle of the instrument in his right hand with the convex border of cannula uppermost and the slot-like terminal extremity pointing towards the

Fig. 9.

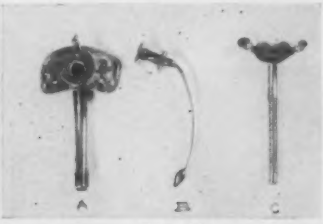

Fig. 10.

Fig. 9. Showing distal end of "Reverse Tracheotome" emerging from tracheotomy wound of patient previously tracheotomized by "orthodox" method. Compare Fig. 6. As the distal end of instrument is being withdrawn into trachea, the tracheotomy tube, held in operator's right hand, is being introduced into tracheotomy opening, the end of tube held against "retreating" end of tracheotome, the latter being used as a guide. (Patient obtained through kindness of Dr. R. Gover, of Willard Parker Hospital).

Fig. 10. Special tracheotomy tube set for experimental work upon goose or dog. (a) Outer tracheotomy tube. (b) Introducing obturator. (c) Flexible spiral metal inner tube.

patient's mouth (Figs. 3 and 7), which is held open by a mouth gag previously inserted.

The operator's left index finger is quickly passed backwards over the dorsum of patient's tongue and hooked over the upper border of the epiglottis, fixing the larynx, and much in the manner in which one introduces an O'Dwyer intubation tube, the flattened end of the curved cannula is forced between the vocal cords, the patient's head at the same time carried slightly backwards (extended) (Fig. 4) until one notes the bulging anteriorly in the midline of the neck (Figs. 5 and 7), caused by the pressure of the distal end of the cannula against the anterior wall of the trachea and pretracheal tissues. In cases not of an emergency character, after preliminary cocainization of the larynx, one may introduce the tip of the reverse tracheotomic cannula between the vocal cords and into the trachea, by indi-

rect vision, using a laryngeal mirror and reflected light of a head mirror.

The curve of the cannula in its long axis permits of its end being placed between any of the membranous interspaces of the upper three or four tracheal rings (Fig. 5). Quickly determining the desired point of puncture, and firmly holding the blunt open end of cannula against midline of anterior wall of the trachea, and the head of patient steadied by an assistant, the concealed blade is "pushed home" by pushing the upper button-like end of the shaft with one's right thumb, when the knife point will be seen coming through the midline of the neck anteriorly (Figs. 6 and 8); when the blade is completely pushed through, the cannula is immediately forced through the wound from 1 to 2 c.m. and the inner shaft with knife-like end

Fig. 11.

Fig. 12.

Fig. 11. A. Bare spot on anterior surface of goose's neck. The small dark spot, in the center of which is the opening through the midline of neck into the trachea, produced by a reverse tracheotomy six weeks previously (see Fig. 13) (B).

Fig. 12. Special tracheotomy tube for goose being introduced through tracheotomic opening shown in Fig. 11.

quickly withdrawn from the cannula from above, while the latter remains *in situ*.

There will immediately be an inrush of inspired air, practically no bleeding and no emphysema.

This entire procedure, while taking several minutes to describe, can be performed in less than half a minute. As soon as the patient breathes tranquilly, the curved cannula, with the perforations at its distal end acting as a temporary tracheotomy tube and its large breathing canal between the vocal cords serving as an intubation tube, a proper sized tracheotomy tube is inserted into wound by holding the distal end of its obturator point against the slot-like end of the cannula (Fig. 9), and as the latter is withdrawn, pushing in the tracheotomy tube, then quickly withdrawing the obturator from the latter.

If desired, the tracheotomy opening may be enlarged by means of a specially constructed tracheotomy scissors, the blunt probe-like end of the longer lower blade of which is inserted through the wound into the tracheal lumen and the pretracheal tissues, including the skin, cut as desired in a vertical direction. Any bleeding points thus created may be immediately ligated.

Following the placement of the tracheotomy tube, the cannula is withdrawn from the larynx and mouth; the immediate dressing made, and after-treatment instituted as usual.

Summary: As Gill[5] well summarizes:

"The accidents which may be incident to the orthodox method of tracheotomy are:

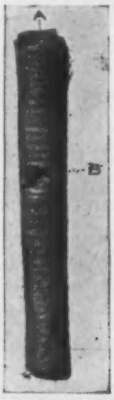

Fig. 13. A. Trachea of goose shown in Fig. 11 removed post-mortem after wearing tracheotomy tube six weeks after reverse tracheotomy was performed. B. Tracheotomic opening showing condition of two tracheal rings six weeks after incision made during reverse tracheotomy.

"*a.* Failure to open the trachea, especially in very fat children.

"*b.* Severe hemorrhage when the incision is carried too far to either side.

"*c.* Irregular incision, making it difficult to introduce cannula.

"*d.* Asphyxiation from blood escaping into the trachea.

"*e.* Asphyxiation from collapse of the trachea.

"*f.* Septic pneumonia or lung abscess caused by the escape of blood and infected material into the trachea and bronchi."

The above-mentioned accidents may all be avoided by the use of the "reverse" tracheotome.

Conclusions: To those who prefer using an intratracheal guide as a preliminary step to performing the classic tracheotomy, as advo-

cated by Lewis[4] and also by Gill[5], I have devised a similarly shaped instrument consisting of a solid curved shaft with a handle at its proximal end, and with a flat, rounded distal extremity, which is introduced into the trachea in the same manner as is the reverse tracheotome; and while the distal end is firmly held against the median line of the anterior wall of the trachea, at the desired point of entry, noted by a bulging anteriorly in the midline of the neck, one cuts down upon the trachea, using the flattened, rounded end of the instrument as a guide.

I do not claim that this method of tracheotomy can or should replace the classic operation in which the trachea is entered from without, but simply desire to call attention to an additional method of performing the operation, which under certain conditions might prove of value.

Whatever merit it possesses will no doubt be determined in the future by those who might be inclined by circumstances to utilize this method.

I have had the privilege of discussing this method both in America and abroad, in France, Italy, Germany and Austria, with many eminent laryngologists; their main criticism was the possibility of entering the esophagus by mistake, but this serious accident could only result from the careless or unskillful use of the reverse tracheotome.

This mishap, however, can be avoided in two ways: *a.* by either passing the instrument between the vocal cords by direct or indirect vision and verifying its presence in the tracheal canal by feeling its distal end between any two of the upper three or four tracheal rings against a membranous intercartilaginous section. If it is in the esophagus the rounded end of the cannula cannot be felt. A little practice on the cadaver or live subject readily acquaints the operator with this method of testing its proper position.

b. Another and very simple method of determining whether the instrument is in the tracheal canal is by placing one's ear near the upper (proximal) opening of the cannula, when one can hear the air coming through the tube as well as feel the expired air against one's face.

It is assumed, excepting in extreme emergencies, that operations of this type are only executed by those skilled and specially trained in their use.

As Gill[5] states: "There are times when all laryngologists are called upon to perform a tracheotomy in cases when intubation will not answer: *a.* When intubation tubes are not available or their use is not understood. *b.* In excessive edema of the larynx when the intu-

bation tube does not relieve. *c.* When the membrane is in the lower tracheal tract."

And it is to meet these indications that I offer this original method of tracheotomy as a substitute for the classic operation.

Animal Experimentation: After several successful trials with the reverse tracheotome on rabbits, dogs and young lambs, the susceptibility of the rabbits to secondary pulmonary infections and the difficulties entailed in the postoperative "nursing" of dogs and lambs, led me to investigate the possibilities of the goose as experimental material. The latter I had already utilized in my study of animal material for bronchoscopic practice.

The study of the trachea of fowls revealed that of the matured goose as having not only the widest tracheal canal but seemingly a greater resistance to secondary infections than chickens, turkeys and ducks.

Thus a large, mature, domestic goose was selected and utilized with success. I have performed the operation successfully on twenty geese, all of which survived until utilized for additional endobronchial experiments, following which they were killed by chloroform previous to postmortem examination.

The last goose tracheotomized by this method is the subject of the present moving picture demonstration. At the end of six weeks, during which period it wore the special tracheotomy tube (Fig. 10), illustrated above, it was in excellent health. I succeeded in producing an experimental pulmonary abscess in its lower left lung, and then hastened its death by chloroform.

Operation on Goose: It is starved for twelve hours previous to operation to prevent regurgitation of food during operation. Day before operation the feathers are dry plucked from the front of the neck for an area 4 c.m. by 7 c.m., about 10 c.m. below the angle of jaw (Fig. 11). The goose is prepared exactly as described above for the human subject, as strict asepsis being maintained as is possible.

The body is covered with a sterile sheet fastened around the neck just below field of operation; the latter is painted with 3 per cent iodine solution, the bills and head are washed with 95 per cent alcohol and mouth, tongue, pharynx and larynx painted with 1-250 acriviolet solution.

The upper surface of larynx is painted with 2 per cent solution of cocain, and by grasping the tongue with a square of sterile gauze and pulling it forward, a laryngeal cocain syringe with narrow curved cannula is introduced into the exposed larynx, and 1½ c.c. of a 1 per cent solution of cocain with 2 minums of 1-1000 solution of epinephrin chlorid is injected into the trachea. After waiting five min-

utes, the "reverse tracheotome", same size and type utilized in human subjects, is used in the same manner as described above under "Operative Technic."

The special tracheotomy tube, however, made for animals must be used as the ordinary human tracheotomy tubes will not fit. The insertion of the tracheotomy tube and the daily dressing must be performed exactly as in human subjects; the same asepsis, care and feeding being required if successful results are desired.

During the first twenty-four hours no food is offered, but a clean pan of water containing a teaspoonful of sodium bicarbonate to the pint should be placed handy. The straw on floor of coop must also be changed daily. With ordinary attention, the tracheotomized goose may live its full life cycle.

After the lung abscess experiment, the trachea was removed at autopsy, a picture of which is presented (Fig. 13), showing a cleanly healed opening in the anterior wall of the trachea (formalin preparation). In several geese operated previously to this one, after one to three weeks the tracheotomy tube was removed, and aseptic dressings applied daily. The tracheotomy wound healed spontaneously and the vocal function returned to its previous normal vigor.

The motion picture demonstration will show the use of the reverse tracheotome on a goose, which was aseptically prepared and anesthetized locally as above described, as well as its demonstration on a manikin and upon two patients previously tracheotomized.

I wish to express my thanks especially to Dr. Chevalier Jackson, of Philadelphia; Dr. John Mackenty and Dr. R. W. Gover, of New York, and other colleagues, respectively, for their kind encouragement and the material afforded for experimental work, which permitted perfection of the instrument and operative technic. The instruments were made by Pilling of Philadelphia.

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125 West 72nd Street.

I. OPEN SAFETY PIN IN THE STOMACH, THEN POINT DOWNWARD IN THE ESOPHAGUS. REMOVAL.*

Dr. Samuel Iglauer, Cincinnati.

II. AUTO-EXPULSION OF FOREIGN BODY FROM THE STOMACH.**

Dr. Henry Boylan Orton, Newark.

When an open safety pin is swallowed, and remains in the esophagus, it is almost an invariable rule to find it lodged with the point upward. On mechanical grounds it is readily understood that for a safety pin to enter the esophagus, the spring coil must usually precede the point of the pin, which otherwise would tend to catch in the esophageal wall. The mechanism closely resembles that involved when a fish swallows the hook. On very rare occasions, however, a safety pin may be found in the esophagus in the reverse position, *i.e.*, with the coil upward and the point downward.

Dr. Jackson in a personal communication has very kindly furnished me with an analysis of his records, as follows: "We have complete records of 164 safety pins removed from the air and food passages. * * * The 164 safety pins were taken from 158 patients, some of the cases having more than one pin; in some instances two, and in other instances three, and in one case, four pins. There were 151 cases in which the point was directed upward. In 13 cases the point was downward, including 10 instances in which the safety pin was point downward in the esopahgus." I have encountered two cases of this kind. (Since this paper was presented, an additional case of open safety pin, point downward, in the esophagus of a child, age 19 months, was referred to me. The pin was successfully removed by my colleague, Dr. Edward King.)

Case 1: Reported in 1922, occurred in the infant daughter of a physician. A Roentgenogram taken on the day of the accident showed this pin open end downward in the lower third of the esophagus. At esophagoscopy curds of milk were found in the lower esophagus. Upon withdrawing the tube the patient regurgi-

*Read before the American Bronchoscopic Society at Atlantic City, May 21, 1927.

**Discussion of the same subject by Dr. Henry Boylan Orton, at the same meeting.

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tated the curds and pin (1 inch long), which fell upon the floor. The parents were certain that the child had not vomited prior to this.

The second case occurred recently. *History:* George G. (L10414), colored, age 7 months, was admitted to Pediatric Service of the Cincinnati General Hospital, Nov. 26, 1926, with a history of having swallowed an open safety pin on the morning of the same day. Fluoroscopy and the Roentgen film showed an open safety pin freely movable in the stomach (Fig. 1). I advised daily X-ray examination to see if the pin would pass through the bowel. Two days later, fluoroscopy and the Roentgenogram showed the pin point downward

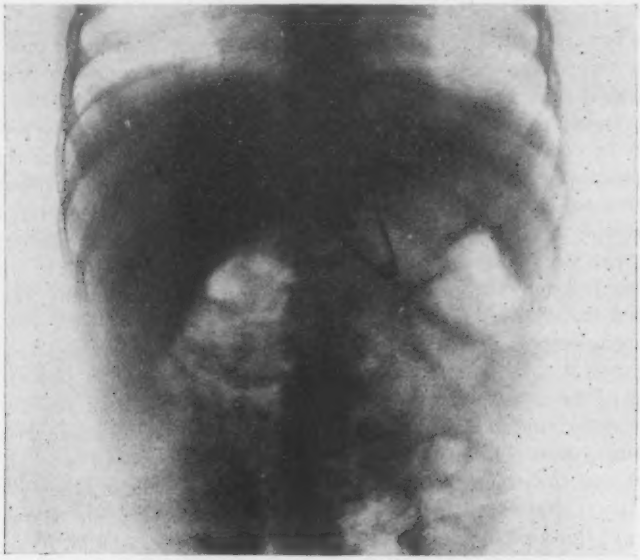

Fig. 1. X-ray film showing open safety pin in the stomach.

in the esophagus on a level with the fourth and fifth dorsal vertebrae (Fig. 2).

Operation: Without anesthesia, a short esophagoscope was introduced. The pin was not seen until the instrument was partially withdrawn, the foreign body having been concealed by a fold of mucous membrane and having been over-ridden by the esophagoscope. The spring coil was seized with a forceps and the pin was pulled into the tube and withdrawn in that position. The pin was of small size, measuring 1 inch in length (2.5 c.m.). There was a febrile

reaction (102.5°) on the following day, but the child made a complete recovery.

Note by Dr. Graeme Mitchell: "There is no evidence of vomiting of food in this case, before the pin was removed. The mother, who was frequently with the child, and the nurses noted no evidences of gagging, although the child at certain times was not under observation."

(In Case 1 also the parents were certain that the child had not vomited after swallowing the pin.)

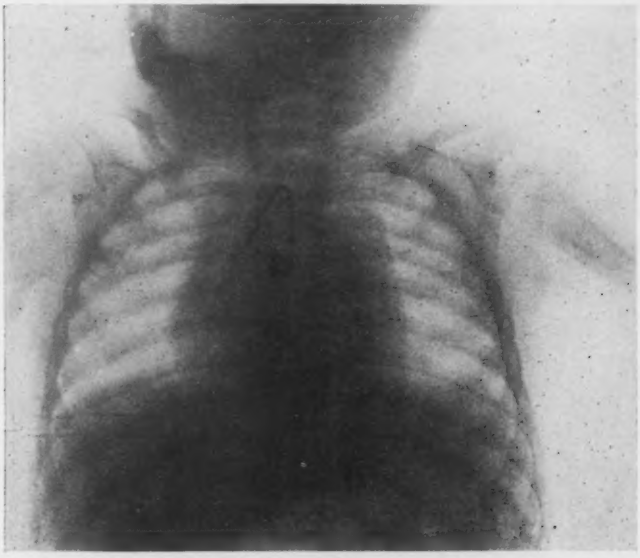

Fig. 2. Roentgenogram taken two days later, showing the same safety pin, point downward, in the esophagus.

DISCUSSION.

Several theories have been advanced to explain the presence of an open safety pin, point downward, in the esophagus. It is conceivable that a small safety pin might enter the esophagus in the usual manner and subsequently undergo spontaneous rotation through 180°. As I have suggested[1], it is also conceivable that a very small pin with a dull point might enter the esophagus with the point downward. It is scarcely justifiable to assume that a pin could be swallowed closed and subsequently open in the esophagus.

Manges was probably the first to suggest that an open pin might enter the stomach, from which it might subsequently be expelled by regurgitation and lodge in the esophagus, point downward. In a recent personal communication and publication, he writes:[2]

"The open safety pin is almost as common as the coin, and they vary in size from quite small to very large safety pins. The open end is usually upward, but in several instances the open end was downward. In one of these, the pin probably opened after it had been swallowed—at any rate, the pin was bent and the point was partly missing, so that its closure was insecure. In another instance, the

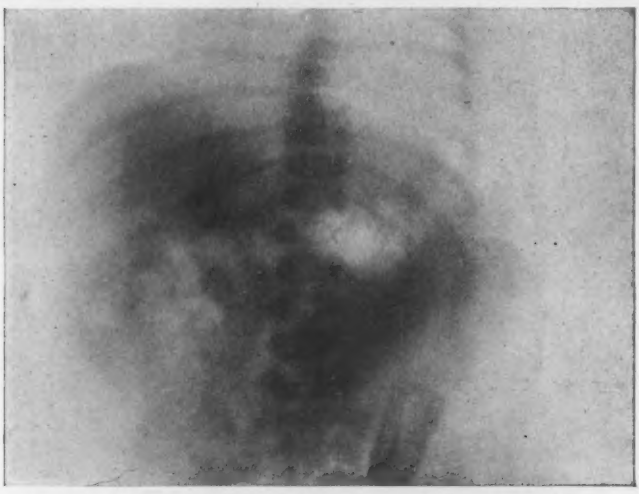

Fig. 3. X-ray picture showing open safety pin in stomach.

safety pin was regurgitated from the stomach into the esophagus, with the open end downward."

This pin had been in the stomach for seven weeks when vomiting attacks carried it into the esophagus, from which it was removed by Jackson.[3]

Comment: Case 2, reported above, confirms the contention of Manges that a safety pin found point downward in the esophagus may have come into this position after having been regurgitated from the stomach.

This, I believe, is the second case on record in which an open safety pin was actually seen (X-ray) in the stomach and subsequently was observed point downward in the esophagus, as described.

In neither of my cases, both of which were closely observed, was there any history of actual vomiting. There may have been simple regurgitation without vomiting.

Under the title of cardioesophageal relaxation, Robins and Jenkelson[4] have recently described the occasional occurrence of reflux of the gastric contents into the esopahgus. Presupposing such an abnormally relaxed orifice, a foreign body might, on rare occasions, be carried from the stomach into the esophagus.

AUTO-EXPULSION OF FOREIGN BODY FROM THE STOMACH.

The rarity of this condition I think justifies a report. As far as I am able to ascertain there has been no case on record of an open

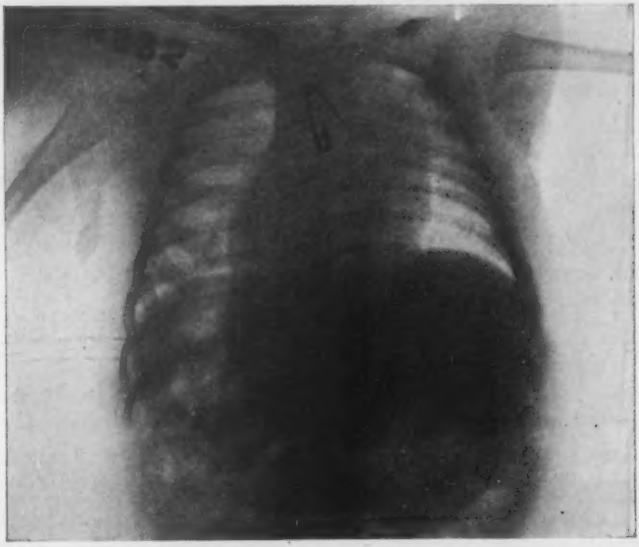

Fig. 4. X-ray picture showing open safety pin, point down and to the left in the esophagus.

safety pin which was swallowed, lodged in the stomach, then regurgitated into the esophagus and lodged there, later spontaneously regurgitated out of the mouth, falling into the hand of the mother.

At the recent meeting of the American Bronchoscopic Society in Atlantic City, Dr. Iglauer, of Cincinnati, reported a case of an open safety pin in the stomach; regurgitated into the esophagus and then removed by esophagoscopy.

The report of my case is as follows: Baby E. G., age 7 months, while in her crib on March 18, 1927, managed to loosen and remove a safety pin which was in her sweater. She apparently placed the pin in her mouth and swallowed it. The mother noticed the child choking and a little cyanotic, and being apprehensive of the child having swallowed something, immediately took the child to a doctor, who suggested to the mother that she take the child to the city nospital, where X-rays would be taken.

X-ray (Fig. 3) was taken at the Newark City Hospital the afternoon of March 18, 1927, and shows the open safety pin in the child's stomach. The interne at the hospital then informed the mother that as the safety pin was in the stomach there was no need to worry as the majority of foreign bodies that reached the stomach passed on, and he instructed the mother to watch the stool for the recovery of the pin and if not found within a week to report back to the hospital for further X-ray study.

The mother, not having recovered the foreign body during the following week, took the child to the hospital on March 25 for another picture. The child during this time apparently had not been disturbed by the lodgment of the pin.

X-ray (Fig. 4), taken on March 25, shows the open safety pin point down and to the left in the esophagus, the spring at about the level of the second costal cartilage.

The patient was then admitted to the hospital and I was notified. The physical examination made at the time of admittance to the hospital was that of a well developed and nourished white baby, apparently in no distress.

Scalp: Negative. Eyes: Pupils equal, regular, react to light and accommodation, no nystagmus, no strabismus. Ears and nose: normal. No rigidity of the neck. Mouth: Negative. Lungs: No dullness, or rales, or modified breath sounds. Heart sounds of good quality and no murmurs. Abdomen: No tenderness or spasm. Extremities: Negative. Reflex: Negative. Skin: No rash of any description.

After seeing the patient and concurring with all the above findings arrangements were being made to transfer the patient to another hospital, where proper facilities were at hand for esophagoscpy.

Just after these arrangements had been made, while in the arms of the mother, the child vomited and regurgitated the pin out of the esophagus, the pin being caught in the mother's hand.

Fig. 4 shows X-ray picture taken after the pin was regurgitated out of the body.

This, as stated before, is the first case of which I know in which an open safety pin has been swallowed, remained in the stomach a few days, then spontaneously regurgitated into the esophagus, remaining at that point for a few days longer, and then regurgitated out of the body.

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FIRST INTERNATIONAL CONGRESS OF OTO-RHINO-LARYNGOLOGY.

The Congress will be held in Copenhagen from July 29th to August 1st, 1928, under the presidency of Professor Schmiegelow. The following subjects have been arranged for general discussion:

1. "The Modified Radical Treatment of Middle-ear Suppuration," introduced by Professor Tapia (Madrid) and Professor Neumann (Vienna).

2. "Septic Diseases Originating from the Throat," introduced by Professor Ferrari (Rome) and Dr. Uffenorde (Greifswald).

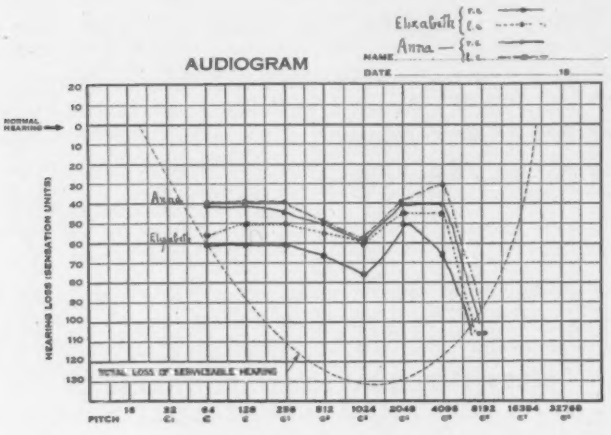
3. "Surgical Diathermy of Malignant Growths in the Upper Air Passages," introduced by Dr. Dan McKenzie (London) and Professor Gunnar Holmgren (Stockholm).

4. "The Anatomical Structure of the Ear and Its Influence on the Course of Suppuration of the Middle Ear," introduced by Professor Mouret (Montpellier) and Professor Wittmaack (Jena).

IDENTICAL HEARING IN IDENTICAL TWINS.

DR. DOUGLAS MACFARLAN, Philadelphia.

The Misses Anna and Elizabeth M. are middle-aged twin sisters who are teaching school. So identical in every respect are these twins that it would be rather a surprise to find the slightest difference between them. They look alike, they are the same height and weight, they act alike, think alike and dress alike. Each had two moles on the face; the moles were identical in size and in location. One sister, Elizabeth, complained of a little stuffiness in the right ear, due



no doubt to a recent head catarrh. For curiosity sake the hearing of each was taken with the 2-A Western Electric audiometer. The interesting parallelism as shown in the audiograms is very evident; the hearing rises and falls across the pitch range in nearly exact correspondence one with the other. Further than this the individual ears are strangely alike in hearing. The difference in level in Elizabeth's audiogram is due to her recent catarrh. Neither of the twins has had previous ear trouble of any moment.

1805 Chestnut Street.

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BOOK REVIEWS.

Stuttering and Associated Aphasia. By Dr. Emil Fröschels. With 12 illustrations in text. Leipzig and Vienna: Franz Deuticke.

This monograph of 134 pages presents in comprehensive form a clear survey of this form of speech defect and includes practical pedagogic exercises and methods of correction of this defect in its many phases.

The author has had a wide experience both as a laryngologist and as a teacher, and the methods and deductions as presented in this little volume are sound and clear. M. A. G.

Contributions to the Art and Science of Otology, Lectures and Papers. By Richard Lake, F.R.C.S.; G. E. Duveen Lecturer on Otology, University of London; Surgeon in Ear, Nose and Throat Department, University College Hospital, etc. London: Macmillan and Co., Limited, St. Martin's Street. 1926.

This volume consists of contributions of lectures and papers in Otology, issued from 1892 to 1925 and revised to keep abreast of the progress in this specialty.

We agree with the author that the advantage of such a collection of papers appears to be "that the subject treated receives more attention than is possible in a textbook."

In this volume are included: Structure of the Aural Polypi; A New Method of Dealing with the External Meatus in Operations of the Mastoid; Necrosis of the Osseous Labyrinth—Removal by Operation; Unilateral Aural Vertigo—Removal of the Semicircular Canals; Fixation of the Stapes; Notes on Rinnés Test; Operation on the Vestibule for the Relief of Vertigo—Description of the Flap Employed in Order to Obtain a Better View of the Parts During Operation; Ablation of Both Vestibules for the Relief of Vertigo; Absolute Unilateral Deafness in Children; Early Otosclerosis Treated with Radium; Remarks on So-called Re-education of the Deaf; Blood Pressure—Its Relation to Vertigo and Tinnitus; Otosclerosis—Does It Exist As a Separate Disease?

We have picked out the most salient papers because they express definite and individual observations of the author and, for that reason, are of special interest. M. A. G.

Cancer of the Nose. By G. Portmann, Professor de Clinique of Oto-Rhino-Laryngologique, a la Faculte de Medecine de Bordeaux; and Henri Retrouvey, Oto-Rhino-Laryngologiste des Hospitaux de Bordeaux. One volume, 980 pages with 248 illustrations. Paris: Gastoin and Cie, Editeurs, 8 Place de l'Odeon. 1927. Price 150 fr.

This is an extensive treatise on Cancer of the Nose, the Nasal Fossa, the Accessory Sinuses and the Nasopharynx, illustrated profusely and with unusual clarity. The volume is divided into three sections; (1) Cancer of the Nose; (2) Cancer of the Nasal Fossa and Accessory Sinuses; (3) Cancer of the Nasopharynx. Every phase of Cancer in this area of the body is minutely discussed, including its historical developments, anatomy, clinical diagnosis, neoplasm variations; gross and microscopic pathology; all forms of treatment; operative technique and its many plastic problems; radiograph and radiotherapy; X-ray treatment; accidents and emergencies and an exhaustive bibliography.

The rich experience and qualifications of the authors enables them to present this important treatise authoritatively and it should be regarded as a very valuable contribution to Rhinology. M. A. G.

Laryngeal Tuberculosis. By Frank Robert Spencer, A.B., M.D., F.A.C.S., Asst. Professor of Otolaryngology, Department of Medicine and Surgery, University of Colorado; Otolaryngologist, University of Colorado General Hospital, and Chief of Ear, Nose and Throat Service, July 1 to September 30; Fellow, American Otological Society, etc. With a chapter on Gross Postmortem and Microscopic Pathology by Philip Hillkowitz, B.S., M.D., Director of Clinical Laboratories, Mercy Hospital, St. Anthony's Hospital and Beth Israel Hospital, New York; Consulting Pathologist, Denver General Hospital and Sanatorium of the Jewish Consumptives' Relief Society; formerly Professor of Pathology, Denver and Gross College of Medicine, etc. Illustrations. Philadelphia and London: J. B. Lippincott Company, 227 South Sixth Street. 1927.

This is an interesting monograph of 70 pages, covering many practical aspects of the subject and includes seven pages of colored plates of Pathological Changes in the Larynx and numerous drawings in black and white; all of the original drawings were made by the use of indirect laryngoscopy.

Chapters on Differential Diagnosis of Laryngeal Tuberculosis; Technique of Cauterization by the Direct or Indirect Method; Discussion of Suspension Laryngoscopy in the Treatment of Laryngeal Tuberculosis; Heliotherapy; the Solar Laryngoscope; Radium and Roentgen-ray Therapy and a chapter on Gross Postmortem and Microscopic Pathology, by Dr. Philip Hillkowitz are included in this monograph and the concluding bibliography on Laryngeal Tuberculosis is fairly complete.

M. A. G.

Plastic Surgery of the Head, Face and Neck. By H. Lyons Hunt, M.D., L.R.C.S. (Edin.), Licentiate of the Faculty of Physicians and Surgeons of Glasgow; late Captain M.C., U. S. Army; Consulting Plastic Surgeon, Midtown Hospital, New York; Consulting Plastic Surgeon, Lexington Hospital, New York; Fellow, American Medical Association. Illustrated with 342 Engravings and 10 Colored Plates. Philadelphia: Lea & Febiger, South Washington Square. 1926. Price $7.00 net.

Plastic Surgery of the Head, Face and Neck since the World War has developed into a special domain as contra-distinguished from General Surgery and the demand for this highly specialized technique has developed many new ideas in the disposal of cosmetic defects and deformities and has called forth originality of the technical skill of the surgical craftsman. The initial chapter is an historical and chronological review of Plastic Surgery that always fascinates the interested reader.

In his chapter on General Considerations the author aptly refers to the numerous experiences of World War surgeons and the tremendous demand for training in Plastic Surgery. "This demand was successfully met by the War Department creating hospitals for the treatment of face injuries and disfigurements. To these hospitals oral surgeons, aural surgeons, rhinologists, dental surgeons, ophthalmological surgeons and brain surgeons were ordered. The reconstructive work was most interesting. Plastic Surgery became fathered by specialists in all the most intricate and difficult forms of head surgery. These men were really the fathers of modern Plastic Surgery; a new, a worthy and a difficult specialty was created."

The book contains chapters on Prosthesis, Grafts and Transplants, Wounds, Keloids, Congenital Superficial Defects, Burns, Scalp and Skull Defects, Repair of Defects in the Region of the Eye, Otoplasty, Rhinoplasty, Meloplasty, Fractures and Defects of the Maxilla and Mandible, Harelip and Cleft Palate, Cheiloplasty, Stomatoplasty, Repair of Defects of the Cervical Region, and a concluding chapter on Physiotherapy in Superficial Surgery of the Face.

This volume is a vade mecum of ingenius devices and suggestions in surgical repair technique and is worthy of the study of every Head Surgeon.

M. A. G.

Practical Otology. By Morris Levine, M.D., Associate Professor of Otology, New York Post-Graduate Medical School and Hospital; Associate Attending Otologist, New York Post-Graduate Medical School and Hospital. Illustrated with 145 Engravings and 3 Colored Plates. Philadelphia: Lea & Febiger, S. Washington Square, 1927. Price $5.50.

This book represents a compilation of Lectures on Otology given at the New York Post-Graduate Medical School and Hospital during the past twelve years and was written primarily in response to the demand from the students of that institution that a volume be prepared embodying the substance of these clinical lectures.

It contains, mainly, the individual experiences of the author in twenty years of otological practice and is intended to reach three classes of readers: The post-graduate student in otology; the under-graduate in medicine, and the general practitioner.

Otological treatment has been stressed; the relation of the general condition of the patient to the otological findings has been emphasized; there are many original illustrations, including three colored plates of special merit.

The chapters on Septic Sinus Thrombosis, Operations for Septic Sinus Thrombosis, Diseases of the Middle Ear, Operations on the Labyrinth, Abscess of the Brain, and a final chapter on Tumors of the Acoustic Nerve round out in very concrete form an admirable manual. M. A. G.

Nasal Neurology, Headaches and Eye Disorders. By Greenfield Sluder, Clinical Professor and Director of the Department of Oto-Laryngology, Washington University School of Medicine, St. Louis. With 167 Illustrations, including 2 Color Plates. St. Louis: The C. V. Mosby Company, 3525 Pine Blvd. 1927. Price $11.50.

The Neurological phase of Rhinology has developed into a factor of much importance and the original research and careful study of the author concerning some Headaches and Eye Disorders of Nasal Origin has led to the elaboration of the present monograph. Special prominence is given to the discussion of the Involuntary Nervous System which is superficial in the nose and nowhere else in the body.

Some of the special chapters are: Vacuum Frontal Headaches with Eye Symptoms Only; Anterior Ethmoidal Neuralgia; The Syndrome of Sphenopalatine Ganglion Neurosis; Hyperplastic Sphenoiditis and Its Clinical Relation to the Environing Nerves.

As a pioneer in Neurorhinology the author has specially qualified by his many years of experience and study in the presentation of this unusual and important monograph. M. A. G.

Nasal Accessory Sinuses. By Professor Dr. M. Hajek, Chief of the Laryngo-Rhino-Otological Clinic, University of Vienna; translated and edited by Joseph D. Heitger, A.B., M.D., Louisville, Kentucky; and French K. Hansel, M.D., M.S., St. Louis, Missouri. Fifth edition completely revised, Volumes I and II. St. Louis: The C. V. Mosby Company, 3525 Pine Blvd. 1926. Price $17.00.

In the preface to the American edition, the editors of this monumental treatise on the Nasal Accessory Sinuses write: "Professor Hajek's work, Pathologie and Therapie der entzundlichen Erkrankungen der Nebenhöhlen der Nase, now in its fifth edition, which embraces the experiences, observations and views of the world's greatest authorities, as well as much original work of the author, extending over a period of nearly forty years, is considered a classic on the Continent of Europe.

The first edition, which was published in 1898, to satisfy the insistent demand for the reproduction in book form of "Hajek's Famous Courses," placed the

anatomy, pathology and therapeusis of suppurative affections of the nasal accessory sinuses, for the first time, on a solid foundation. In the subsequent editions a most significant and impressive feature is that Dr. Hajek found it unnecessary to retract or modify a single basic principle originally presented.

As there is much diversity of opinion on matters pertaining to the Nasal Accessory Sinuses, this translation offered to English-speaking Rhinologists will serve to develop a better understanding, standardization and unification of widely divergent views regarding diagnosis and treatment; it will place at the disposal of English readers the original conceptions of this Austrian master in Nasal Accessory Sinus Diseases for differential diagnoses and treatment to a degree not formerly attained.

This valuable treatise, ably edited and translated by Drs. Heitger and Hansel, should be in the library of every Rhinologist for it is an authoritative reference volume in every angle of the Nasal Accessory Sinuses and their important role in Rhinology. M. A. G.

Malattie del Naso e Della Gola. By Professor Salvatore Citelli, Ordinario de Clinica Otorinolaringoiatrica nella R. Universita de Catania. Second edition of 686 pages, 3 Colored Plates and 234 Illustrations throughout the text. Torino: Unione Tipografico-Editrice Torinese. 1926.

Professor Citelli, one of the modern Italian masters in Rhinolaryngology, presents the second edition of his excellent work on Diseases of the Nose and Throat. This volume discusses Semeiology, Anatomy, Clinical Findings, Treatment and Surgical Technique of practically the entire range of Rhinology and Laryngology; discusses rather extensively Diseases of the Accessory Sinuses and Neurosis of the Larynx; emphasizes Ozena, which seems to be more prevalent in Italy than in other Continental European countries or American, and still advocates the Mathieu Tonsillotome in Tonsillectomy; Intubation is still included as a classical chapter; Scarletinal and Typhoid Laryngitis are given dignified consideration and early diagnosis of Laryngeal Carcinoma followed by Laryngotomy and Submucous Dissection and Excision of Neoplasm is advocated.

Esophagoscopy and Bronchoscopy is included and brought up-to-date in this chapter. M. A. G.